

Exhibit 5

PLAINTIFFS' OPPOSITION TO DEFENDANTS' MOTION TO EXCLUDE PLAINTIFFS' EXPERTS' GENERAL CAUSATION OPINIONS FOR FAILURE TO ACCOUNT FOR SECTION 230 AND THE FIRST AMENDMENT

Case No.: 4:22-md-03047-YGR

MDL No. 3047

In Re: Social Media Adolescent Addiction/Personal Injury Products Liability Litigation

HIGHLY CONFIDENTIAL (COMPETITOR)

**UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF CALIFORNIA
OAKLAND DIVISION**

IN RE: SOCIAL MEDIA ADOLESCENT
ADDICTION/PERSONAL INJURY PRODUCTS
LIABILITY LITIGATION

THIS DOCUMENT RELATES TO:

ALL ACTIONS

MDL No. 3047

Civil Case No. 4:22-md-03047-YGR:

Honorable Yvonne Gonzalez Rogers

**EXPERT REPORT OF DR.
MICHAEL BAIOCCHI, PHD**

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HIGHLY CONFIDENTIAL (COMPETITOR)**I. Introduction****a. Qualifications**

1. I am a tenured associate professor in the Department of Epidemiology & Population Health at Stanford University. I have additional appointments in the Department of Statistics and the Department of Medicine. My principal research areas are: (i) causal inference, particularly in estimation of causal effects from observational data, (ii) gender-based violence prevention, and (iii) health outcomes research (e.g., adolescent nicotine use prevention, cardiothoracic interventions).

2. From 2006 to 2011, I trained as a statistician, receiving a Ph.D. from the Wharton School at the University of Pennsylvania. After my graduate studies, I was awarded the Stein Fellowship, one of the top fellowships in the field of statistics, in the Department of Statistics at Stanford University. I began a professorship as part of the Stanford faculty in 2014. Before going to graduate school, I worked at Towers Perrin — an actuarial consulting firm — as an actuarial associate helping to price and procure health insurance coverage plans for clients.

3. A primary focus of my research is developing new, statistically rigorous methods for empirically estimating causal effects. I have designed methods appropriate for estimating causal effects from observational data (e.g., using natural experiments; Baiocchi et al. 2010, Aikens et al. 2020), from randomized controlled trials (Rigdon et al. 2018a, Rosenman et al. 2023a), and data that merges both data from randomized trials and observational data (Rosenman et al. 2023b). I published a highly cited (>750 citations) manuscript on one of the most prominent “natural experiment” study designs (Baiocchi et al. 2014). I have also developed a novel framework for addressing differential measurement error (Rosenman et al. 2023a). I was the senior statistician on a team that discovered and mathematically described a new form of bias (Aikens et al. 2024). Additionally, I have proposed, developed, and published statistical software packages in the programming language R, which allow researchers to implement methods my research team and I have created (Rigdon et al. 2018b, Aikens et al. 2021).

4. In a second line of research, I am the principal investigator on studies of sexual assault prevention. In this line of research, my lab and I have proposed and evaluated two randomized controlled trials of a sexual assault prevention intervention (Baiocchi et al. 2017, Sarnquist et al. 2024). For this line of work, our team has been recognized with the Rosenkranz Award and a Gates Foundation Grand Challenges Grant (Hansen 2024, American Statistical Association 2018).

5. Finally, in another set of research, I evaluate health outcomes and complex policy decisions. I was the lead statistician on an observational study that appeared in the New England Journal of Medicine and was subsequently used to set national guidelines for the treatment of cardiac valve replacement (Goldstone et al. 2017, Otto et al. 2021). My research has used complex natural experiment designs to analyze major health system policies, with results published in the British Medical Journal (Marafino et al. 2021). I have also been the lead statistician on studies of adolescent e-cigarette use (McKelvey et al. 2018).

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6. To summarize, I am a statistician with particular expertise in causal inference, having developed methodologies for rigorously estimating causal effects from both observational data and randomized controlled trials (RCTs). In addition to developing statistical methods, I have also published numerous peer-reviewed papers estimating causal effects from observational data sets. I have also designed and run randomized controlled trials of behavioral interventions. Given my areas of expertise, I limit the scope of my opinion to addressing issues with empirical causal inferences in Professor Jean Twenge's May 16, 2025 expert report, served by the MDL Attorney General, personal injury, and school district Plaintiffs, and the pertinent literature.

7. A copy of my curriculum vitae, which provides further details of my research publications and professional activities, is provided as Appendix A to this report. I have previously testified as an expert witness in the matter *Christina Arlington Smith et al., v. TikTok Inc. et al.*, Judicial Council Coordination Proceeding No. 5255, Case No. 22STCV21355 (Cal. Super. Ct. L.A. Cnty).

b. Engagement

8. I was engaged by counsel for Defendants, who requested I apply my training, expertise, and experience to assess the state of scientific knowledge about whether social media service features cause negative mental health outcomes in adolescents and, in particular, to consider the claims advanced in the May 16, 2025 report submitted by Professor Jean Twenge in this litigation. Further, the focus in Plaintiffs' complaint on specific features of social media services further informs the scope of my opinions — namely, I consider the extent to which Professor Twenge's claims describe specific service features, as isolated from the content they are being used to deliver. All materials considered in reaching my opinions are cited in the References section and in Tables S1 and S2. My opinions are based on my expertise, training, and professional experience in statistics and causal inference.

9. My compensation for my work on this matter is \$1,100 per hour. My compensation is in no way related to the substance of my expert opinions, nor is it contingent on those opinions. During my work on this report, two research assistants worked under my direction and supervision to assist with my literature search and provide research support. All the opinions stated in this report are my own.

c. Summary of Opinions in Response to Professor Twenge's Expert Report

10. After reviewing Professor Twenge's expert report submitted in this litigation, I conclude that the report exhibits a number of key methodological flaws that undermine the validity of Professor Twenge's conclusions. I hold these opinions to a reasonable degree of scientific certainty, and I reserve the right to amend my opinions based on materials, testimonials, and/or other relevant information that may become available, as well as the right to respond to any additional opinions offered by plaintiffs' experts. My conclusions after my review of Professor Twenge's Report are as follows:

Opinion 1: Professor Twenge's causation opinions are not supported by reliable or scientific methodology. Among other fundamental flaws, Professor Twenge improperly

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relies on correlational studies and internal company documents to infer causal conclusions; gives insufficient treatment to findings from systematic reviews and meta-analyses, particularly those that are inconsistent with her causal claims; bases her conclusions on unreliable measures of social media usage and unscientific outcome measures; fails to properly account for confounding factors and biases; and overstates the ability of the cited randomized trials and natural experiments to inform the causal questions at issue.

- **Opinion 2:** Professor Twenge overstates the ability of time series/Granger-causal analyses to inform causal conclusions. Indeed, Professor Twenge misrepresents Granger-causality's relationship with the kind of causality that is at issue in this litigation.
- **Opinion 3:** Professor Twenge's "time series" analysis is methodologically unsound and seriously misstates the ability of time series data to empirically achieve causal inferences.
- **Opinion 4:** Professor Twenge does not offer reliable evidence that the alleged harms can be attributed to the design or features of social media apps, as differentiated from the content they deliver.
- **Opinion 5:** Professor Twenge fails to adequately consider plausible alternative causes for the alleged harms, rendering her causal opinions unreliable.

11. Having assessed all 99 of the citations in Professor Twenge's expert report, I also note that 19 of them were non-peer-reviewed writings (e.g., blog posts, magazines), which would not be considered reliable scientific evidence. There were 36 citations to manuscripts that may be adjacent to the matter under consideration but are not directly material to empirically examining the possibility of a causal connection between social media usage and adolescent mental health in the United States. Of the 44 pertinent peer-reviewed articles Professor Twenge cites:

- 28 of the 44 had study designs that did not allow for assessment of causal claims (e.g., correlational studies).
- Of the 28 that did not have causal study designs, 21 were explicit in stating that their results were non-causal. Of the remaining 10, six were silent on their ability to assess causal effects and two incorrectly claimed they could show causality when their study designs were inadequate.
- Thus, 64% (=28/44) of the academic articles Professor Twenge cites in her claims of establishing causal conclusions were not of sufficient rigor to reliably assess causal claims. 41% (18/44) of the peer-reviewed studies were *explicit* in their own reporting that they are unable to reach causal conclusions.¹

¹ The eight papers that were silent about their ability to reach causal conclusions may have been silent because their authors considered it obvious that the studies were non-causal. Under that interpretation, 59% (=26/44) of the articles were clear on their lack of causal conclusions. In fact, this is how most academics would interpret silence in those eight papers.

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- Only 13 of the 44 employed some kind of experimental design (e.g., randomized trial, quasi-experimental) and thus had a study design that could at least potentially inform causal claims.
- For these 13 studies, even though the studies are designed in a way that could support limited causal inferences, they do not, individually or in the aggregate, show that social media use has a real-world causal connection to adolescent mental health. This is because: (i) they do not measure changes in the kind of outcomes at stake in this litigation (e.g., clinical mental health conditions in adolescents), (ii) they are run under conditions that do not accurately reflect real-world usage of the social media by adolescents, (iii) they do not differentiate between the potential effects of content versus the potential effects features of social media platforms (i.e., they conflate the effect of the content a user is requesting from the platform and the effect of the method by which the platform delivers the content), and (iv) they have not had their results reproduced or had their results replicated under different conditions by different research teams.

12. To visualize the above discussion, I produced Figure A, which also summarizes the types of citations in Professor Twenge's report. The above discussion of the citations in Professor Twenge's report is also summarized below in Table S3.

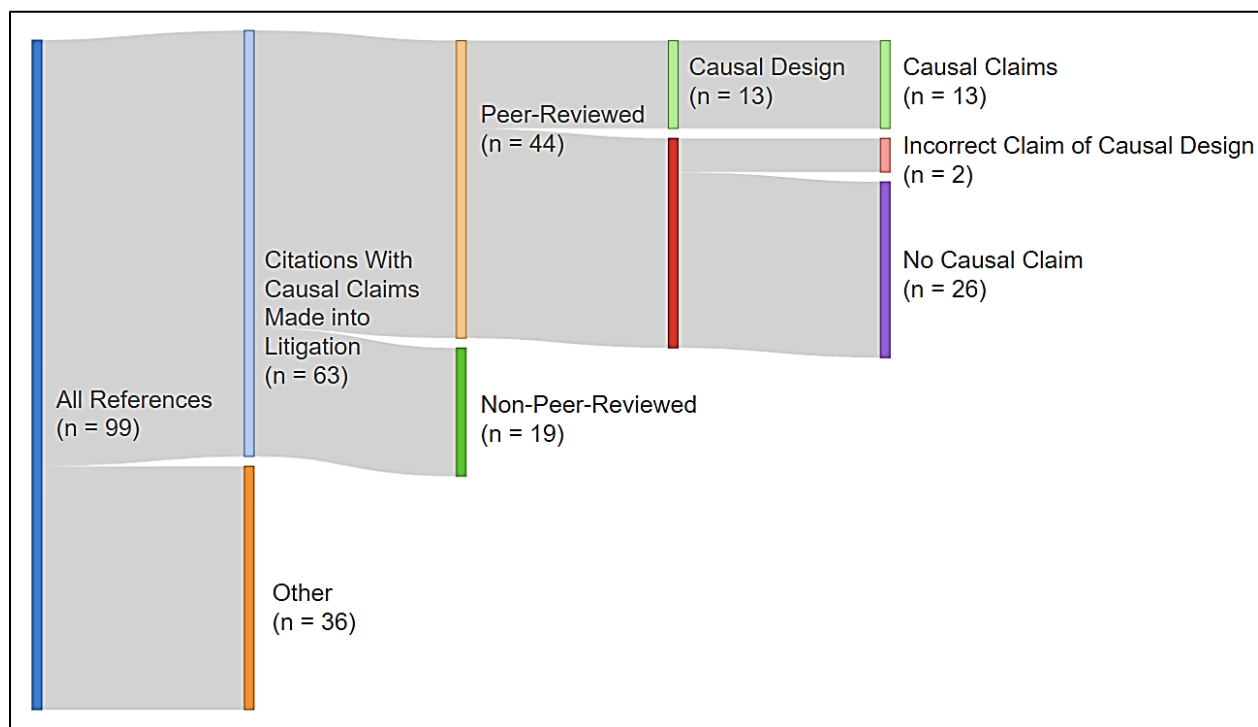


Figure A: Summary of the citations in Professor Twenge's report, labeled by my review. Note that "Other" refers to citations covering topics that may be adjacent to the case but are not material to the empirical assessment of whether a causal connection exists between social media use and adolescent mental health.

13. In addition to my review of the studies Professor Twenge cites, I conducted my own literature search for systematic reviews and meta-analyses of studies on social media use and mental health. In total, I identified 77 peer-reviewed manuscripts, which are documented in

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Tables S1 and S2. My review of these materials further informs my opinions expressed here, as discussed below in Section II.b. My methodology for this search is described below in Exhibit A.

II. Correlational Studies Do Not Provide Evidence of Causation

14. In her report, Professor Twenge significantly overstates the ability of correlational studies to establish causal relationships. In the realm of causal inference, not all study designs are equal, and correlational studies fall near the bottom of the hierarchy.

15. Professor Twenge makes several assertions about correlational studies that give a misleading sense of the rigor of these studies:

- Assertion: [C]orrelation can point toward possible causation. (Page 4, paragraph 20)
 - Response: the issue is not that correlational studies can point towards possible causation, it's that correlational studies indiscriminately point to many possible causes, one of which may be a real cause, but they may also fail to point to any real causal relationship. Correlational studies are not determinative of causal effects; they are only casually related to causality.
- Assertion: Correlational studies are a particularly important piece of evidence in assessing causation when randomized controlled trials are infeasible or unethical. (Page 4, paragraph 20)
 - Response: This is not true. Well designed and implemented natural experiments, which are capable of bounding causal effects, are important evidence in assessing causation when RCTs are infeasible or unethical. But correlational studies are notoriously unreliable for making causal claims, which is why they are considered low-quality evidence.
- Assertion: Correlational studies also often include a much larger number of participants than randomized controlled trials, making them less vulnerable to random variation in results. (Page 4, paragraph 20)
 - Response: Speaking as a statistician, while study sample size can be an issue for some studies, it is rarely the key issue when making evidence-based decisions. The key issue is whether or not a study has bias. We call bias "a first order concern" because bias is of primary importance, whereas measurements of uncertainty (e.g., standard errors or confidence intervals) are a "second order concern." Second order concerns are only material if the first order concerns are addressed. Sample sizes are important, but given that correlational studies are assumed biased until proven otherwise, the sample size of a correlational study is usually only of academic interest. Another way to say this: if an estimate has a very tight confidence interval (is very "precise") but it is around the wrong answer (i.e., "biased") then that is often quite a bit worse than a confidence interval that is appropriately wide and contains the true answer. Correlational studies with large sample sizes are often misleading because they have tight confidence intervals around incorrect causal estimates, this is because they are often biased.

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- Assertion: Correlational studies reveal a dose-response relationship. (Page 5, paragraph 24)
 - Response: Correlational studies are non-causal and a dose-response relationship is a causal concept. This is like saying that the number four is red. Except under very unusual circumstances (e.g., you have grapheme-color synesthesia) these are two different types of things: numbers and colors. Except under very unusual circumstances (e.g., we ran a well-run experiment that randomizes participants to different doses) a correlational summary of data does not reveal a dose-response relationship.

a. Correlational Studies Are Limited in What They Can Show

16. In contrast to correlational studies, randomized controlled trials (RCTs) are valued for their ability to establish causal relationships because — through the careful use of researcher-controlled randomization — they generate data that can be used to estimate a causal relationship in an unbiased way (Cox & Reid 2000). Well-run RCTs are, by design, able to identify changes in the outcome and attribute these changes to a particular set of causes. Their ability to isolate and attribute, and to rule out other potential causes, is why RCTs are so prized in causal inference (Imbens & Rubin 2015, Chapters 1-4 & 12).

17. It is also very much the case that not all randomized controlled trials are well-run or are informative about all related matters. There are many ways in which an RCT might not be well-run, but I focus on three issues common in studies that look at behaviors and/or mental health. Common failure points in a behavioral RCT include: (i) failure of many participants to complete the study (a.k.a., “high dropout”); (ii) “interference effects” (e.g., if one participant in the study interacts with another participant in the study and their outcomes change due to their treatments)²; and (iii) studies in which participants understand what the goals of the study are (e.g., they understand that the researchers believe the intervention will improve mental health), making the participants more likely to distort their responses to surveys to be consistent with the researchers’ goals.³ These are just three very common failures that occur in RCTs that involve humans. RCTs are less likely to have these failures when researchers are studying chemical interactions. RCTs are much more likely to fall short of rigorous causal inference when they involve complex human behaviors and mental states. These kinds of challenges are sometimes referred to as “limiting the internal validity of the study” because they make us believe the estimates are biased and thus doubt the quantitative conclusions.

18. Even if an RCT is well run, it can still be non-informative about related issues. Three common issues can make an RCT non-informative: (i) the RCT uses a sample of

² Consider an RCT looking at the impact of lowering social media use. If one participant was randomized to reduce social media usage but their friend in the study was allowed to use social media and then they discussed “what’s been going on” on social media, then this would potentially bias the study through participants interfering with each other.

³ This challenge is sometimes referred to as “demand characteristics” or “demand effects” of a study.

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convenience (e.g., the RCT uses students from the university where the researchers work, many of whom are taking an introductory psychology class) versus an RCT using a random sample of participants from the population of interest (e.g., adolescents in the United States, including people from all different walks of life and geographically spread out); (ii) the RCT uses metrics that are not aligned with the broader question at hand (e.g., the RCT measures participants' affect — colloquially what we might call “mood” — whereas the important question is really about mental health; or the RCT asks about “happiness,” but we are interested in depression); and (iii) the RCT might not be “ecologically valid” in the sense that the scenario the study puts participants through is artificial and dissimilar to the way people naturally act or interact. (E.g., some RCTs in the social media literature put people into fMRI machines during the study, which is not how most people use social media; other RCTs restrict which features participants can/cannot access or how long they must interact with social media, or have participants interact with actors playing a role, and this artificiality tends to diverge from how people actually interact with friends on social media). These kinds of challenges are sometimes referred to as “limiting the external validity of the study,” because they make us believe the estimates are not representative of the effects we would witness “in the real world.”

19. While it can be hard to construct and implement well-run RCTs that are valid for understanding real-world effects, it is even harder to construct correlational studies that are useful for understanding causal effects. Correlational studies⁴ merely show that two variables are associated (i.e., when one variable changes, the other usually changes as well). It is a basic principle of causal inference and statistics that, unlike RCTs, correlational studies are presumed to have data that will produce biased estimates of a causal relationship (Imbens & Rubin 2015, Chapters 12 & 21). This is important because a researcher cannot eliminate this bias simply by amassing more data. This means that having a larger dataset with a large sample size does not eliminate the bias intrinsic in this type of data.⁵ Having tighter confidence intervals around an

⁴ A note about terminology: a full taxonomy of all kinds of studies is beyond this report's scope. But in these discussions we are using “correlational” and “cross-sectional” to describe studies. While these two terms are not interchangeable, they overlap quite a bit in the sense that the vast majority of studies that use cross-sectional data are limited to producing correlational analyses. “Correlational analyses” are just slightly above “descriptive” in their sophistication. Correlational analyses summarize how two or more variables “appear to move together in the data.” I point this out because studies that use time series data, longitudinal data, or cross-sectional data can be correlational. But, again, nearly all cross-sectional data are limited in what is statistically achievable — producing mostly just descriptive or correlational analyses.

⁵ Professor Twenge uses sample size differences to dismiss longitudinal studies that found no effects: “Most longitudinal studies that do not find effects have smaller sample sizes than the studies finding effects. For example, Heffer et al. (2019) included only 594 individuals, Coyne et al. (2019) included only 500, and Mitev et al. (2021) included only 116 in one sample and 120 in the other, compared to sample sizes in the thousands for those studies discussed above.” (pg. 24). As mentioned above, changing the sample size of a correlational study does not make it more causal or less biased. It merely changes confidence intervals. In fact, large correlational studies

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inaccurate answer doesn't make it more accurate. While statistical fluctuations due to randomness can sometimes be diminished by increasing the amount of data available (Le Cam 1986), if several correlational studies are biased, then "averaging them together" does not produce an unbiased estimate of a causal effect (Kaplan et al. 2014, Sterne et al 2001). This is problematic for meta-analyses of correlational data, even the few that Twenge cites (i.e Liu et al., 2022; Ferguson 2025; Saiphoo et al. 2019; Vázquez-Vázquez et al. 2024).

20. In fact, the empirical academic departments I am familiar with (e.g., epidemiology, economics, political science, biostatistics, statistics, psychometrics) spend 1-2 years training their Ph.D. students in rigorous methods for estimating causal effects, if the students are going to use observational data. This is because, with observational data, there are many variables that are causing the outcome to change — identifying and isolating the impact of one cause (i.e., the potential cause we are interested in) requires careful and rigorous methods. Students are explicitly trained to use methods that surpass the naive and biased correlational study design, because correlational study designs completely ignore the complexity of the real world. Correlational studies are, at best, descriptions of what appears in the world and can be observed by researchers. But, in the complex real world, appearances are deceiving. Correlations invite creative people to conjecture about the potential for causal connections between variables. But these conjectures are not reliable. In academia we have to constantly remind ourselves and our students about the allure of correlational studies.

21. In her report, Professor Twenge's discussion of correlational analyses in her report fails to acknowledge the fundamental and well-known limitations of correlational studies.

b. Professor Twenge Largely Ignores Systematic Reviews, Meta-Analyses, and Consensus Reports⁶

22. Furthermore, Professor Twenge fails to report on the consensus described in systematic reviews and meta-analyses of the literature on social media use and adolescent mental health. These are analyses that take into account many studies and produce synthesized understandings of the overall literature's empirical findings. Only four of Professor Twenge's references are to systematic reviews or meta-analyses (i.e., Liu et al., 2022; Ferguson 2025;

often mislead by having very tight confidence intervals (implying they are quite certain) but are tight around the wrong answer (i.e., they are biased). **Larger data sets do not imply a correlational study has somehow increased the causality of the conclusions.**

⁶ Meta-analyses aggregate many studies that consider the same question and aim to identify quantitative trends across the studies. Systematic reviews are generally an organized summary of the literature, usually in a specific time frame or for a specific set of metrics. Consensus reports provide comprehensive examinations of the literature authored by an expert panel.

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Saiphoo et al. 2019; Vázquez-Vázquez et al. 2024)⁷, despite there being many in this space. To explore this gap, I report on my review of these summative reports.

23. In my review of the literature, I have focused on systematic reviews and meta-analyses because these summaries of the literature are where replications of studies — one of the strongest factors in assessing causality — are noted, where overall methodological rigor is assessed and summarized, and where experts point to additional lines of research needed. If a literature has rigorously established causal connections, then such connections will show up in systematic reviews and meta-analyses. While any particular study may be strong and informative, from a causal inference and statistical perspective, no one study can be deemed to establish a causal connection for a population. A particularly well-run study will need to be replicated by other researchers, with different study participants, in other settings before we typically agree that a causal relationship has been established (Murad et al., 2014). As an example of the role of these summative reports in the literature, the National Academy of Sciences *ad hoc* committee relied upon systematic reviews and meta-analyses in its report on the relationship between social media use and mental health in adolescents.

24. Importantly, systematic reviews and meta-analyses are limited in their value by the underlying studies they consider—*i.e.*, a meta-analysis of cross-sectional data is limited in its ability to reliably establish a causal link (“garbage in, garbage out” is the shorthand used for this dynamic of poor-quality inputs being responsible for poor quality outputs). There are some circumstances where systematic reviews and meta-analyses can identify biases in the articles being analyzed and then help produce better quality insights. However, (i) in my review of the systematic reviews and meta-analyses investigating the links between social media usage and adolescent mental health, I did not see any such methods implemented (and thus no increased ability to reliably estimate causal links), and (ii) there are no citations in Professor Twenge’s report that use these methods for improved causal inference in systematic reviews and meta-analyses.

25. In my literature search for systematic reviews and meta-analyses of social media use and mental health,⁸ I identified 77 peer-reviewed manuscripts. Reflecting the state of the literature on social media and adolescent mental health, nearly all of the studies analyzed in these 77 manuscripts were cross-sectional/correlational studies, with only one manuscript exclusively summarizing the results of only randomized controlled trials. I document these manuscripts in Tables S1 and S2. Of these 77 peer-reviewed manuscripts, only five manuscripts asserted some level of causality (McComb et al. (2023), Liu et al. (2025), Ferguson (2025), Karim et al. (2020), and Memon et al. (2018)), with 69 studies having explicit statements that causality was not established. The remaining 3 manuscripts (Cheng et al. (2024), Gioia et al. (2025), Marciano et al. (2024)) include statements from authors that suggest they believe their results are only “correlational” or only find “associations” but are not explicit about this belief.

26. Two of the five summative reviews that claim causation was established — Karim et al. (2020), and Memon et al. (2018) — are of exceedingly poor quality and would be

⁷ Note that Vázquez-Vázquez et al. 2024 is a systematic review that is not about social media.

⁸ I report the procedure and search terms used for this search in Exhibit A.

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dismissed as unreliable by a reasonable academic. Ferguson (2025) reviews studies that used causal designs and concludes there is little consistent evidence for causal connections between social media use and adolescent mental health in the population (e.g., “Nonetheless, meta-analytic evidence for causal effects was statistically no different than zero”).

27. The McComb et al. (2023) study, while claiming causal conclusions, does not offer much support for that claim because the reported effect sizes in the underlying studies are small, and there is a reported mix of both beneficial and harmful effects. Moreover, the study does not focus on clinically recognized outcomes — the focus of the research summary is on social comparisons, and not on specific conditions like depression or anxiety.

28. Another systematic review I identified that draws causal conclusions is Liu et al. (2025). The paper reviews 20 RCTs (which reported 56 effects) investigating the relationship between social media detox (SMD) interventions and well-being. This meta-analysis exhibits several features that reduce the reliability and usefulness of their conclusions. First, the journal in which it was published has been graded a level 0 and is part of a problematic publishing group (MDPI), and most professionals would be hesitant to rely on a paper from this kind of journal. Second, a primary conclusion from the authors was that “there was heterogeneity in the relationship between SMD and well-being caused by cultural background.” This was a correlational analysis, and therefore “caused” is not a valid term. Third, three of the 20 randomized trials the authors claim to be about “social media detox” are actually about smartphone use in general. 12 of the 56 (20%) effects analyzed came from these three studies. This is a deceptive and confused conflation of distinct exposures (that is, reducing use of smartphones is not the same thing as reducing use of social media), but the authors of this study do not seem to recognize this. Fourth, none of the underlying studies isolates the effect of “social media detox” from the effect of content.

29. The vast majority of systematic reviews and meta-analyses that examine social media use and mental health report that cross-sectional and other studies in this literature show only weak and inconsistent associations; in short, the studies fall well short of establishing causation (e.g., Purba et al. 2023, Fassi et al. 2024, Ansari et al. 2024, Plackett et al. 2023, Valkenburg et al., 2022; Orben 2020). Similarly, the 2023 consensus report issued by an ad hoc committee of the National Academy of Sciences reviewed the correlational, longitudinal, and experimental literature on social media and mental health outcomes in adolescents and concluded that the literature “did not support the conclusion that social media causes changes in adolescent health at the population level.” (Page 92).

30. Large-scale reviews are important to consider when trying to understand if the correlational studies have somehow established causation because there are statistical methods that allow researchers to “triangulate” several biased studies and reach defensible conclusions of causation. Triangulation studies, which use these techniques, often appear in systematic reviews and meta-analyses of the literature, where analysts synthesize the empirical results of many studies. None of the meta-analyses or systematic reviews I read used triangulation techniques or similarly sophisticated methods for assessing the impact of biases to improve causal estimation. The fact that Professor Twenge avoids addressing the consensus reached by the systematic reviews and meta-analyses is concerning because that is where triangulation studies, or similarly rigorous methods for aggregating evidence from correlational studies, would be reported.

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Moreover, the conclusions of these studies are directly at odds with Professor Twenge's claims regarding causation.

c. Trends Other Than Social Media Use Are Correlated with Changes in Adolescent Mental Health

31. To demonstrate why correlational studies like those relied upon by Professor Twenge do not carry the weight of causation in the way she implies in her report, I share a few examples of similarly strong correlations, drawn from the same data set Professor Twenge uses.

32. One of the empirical shortcomings in the figures Professor Twenge presents in her report is that in Figures 1-10, these population-level time series plots do not show the correlation of interest. That is, these plots all omit the usage data of adolescents; there is no time series plot of the amount of usage running alongside the outcomes of interest (e.g., depression, emergency room visits). There is an implied correlation, since the post-2010 period is when we are told that rates were increasing. But it is a notable gap that the actual "correlation" that is at issue is not directly plotted. Figures 1-10 require imagination and credulity since we are not provided the usage information.

33. In the following plots, I follow Professor Twenge's convention. I do, though, overlay additional time series to show how strongly they correlate with self-reported depression rates.

34. In Figure B, I use data from the Youth Risk Behavior Surveillance Survey (YRBSS), which is a data set Professor Twenge uses to identify correlations between social media use and depression, to plot the rise in adolescents who report not eating breakfast against the rise in rates of adolescents who report depression. Figure B offers a visualization of strong correlations. As discussed above, a correlation does not provide strong enough evidence for a causal argument, so these correlations do not show that an increase in missing breakfast *caused* increases in depression (or that social media services caused breakfast eating to decrease). The real issue here is that correlations are often just coincidences, rather than causal facts. With the vast amount of data available today on global phenomena, it's easy to find coincidental correlations if we look hard enough.

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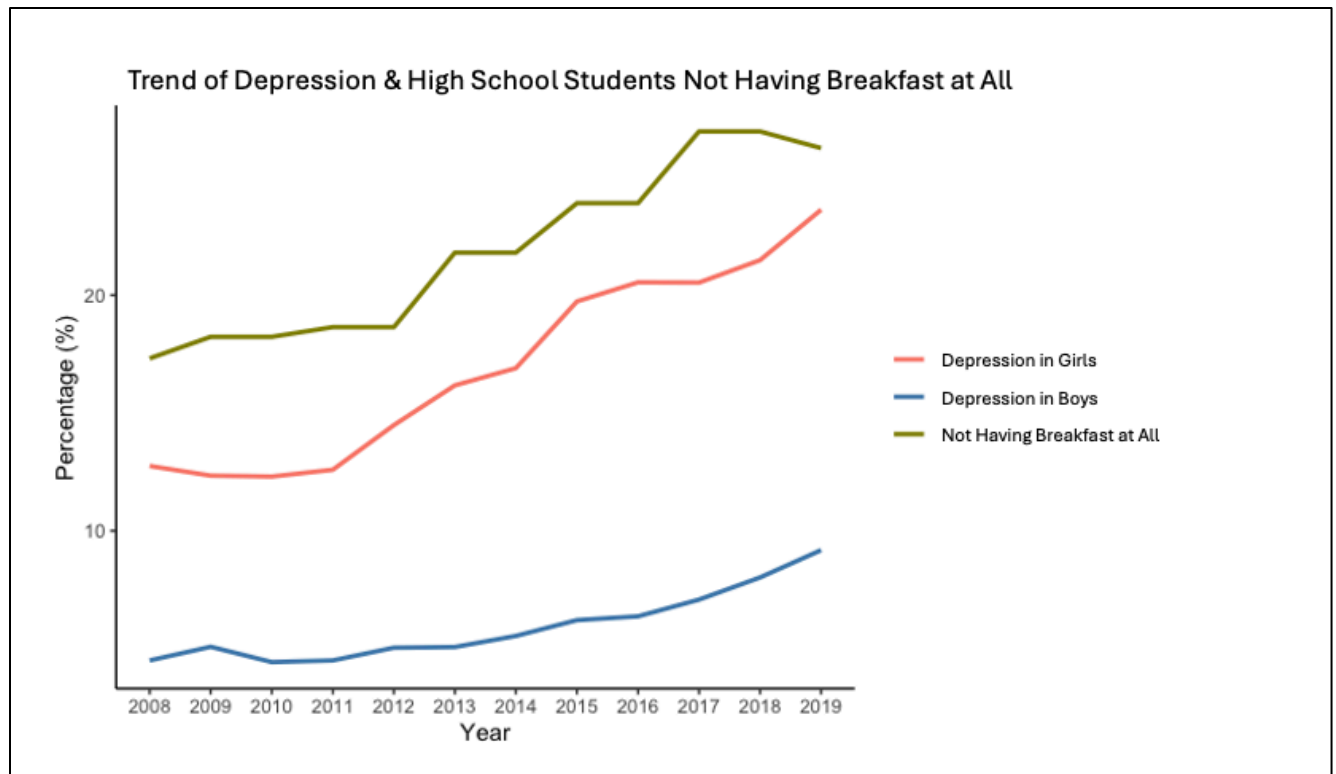


Figure B: plotted using data from the YRBSS

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35. Next, at the same time we saw self-reports of depression and social media use increasing in adolescents, the YRBSS data set showed that the rate of adolescents who had ever had sex was decreasing. I present this in Figure C. There is a strong (negative) correlation in Figure C, but this correlation is likely due to a complex mix of many other factors, and it does not show that a decrease in sex caused an increase in depression (or an increase in the use of social media).

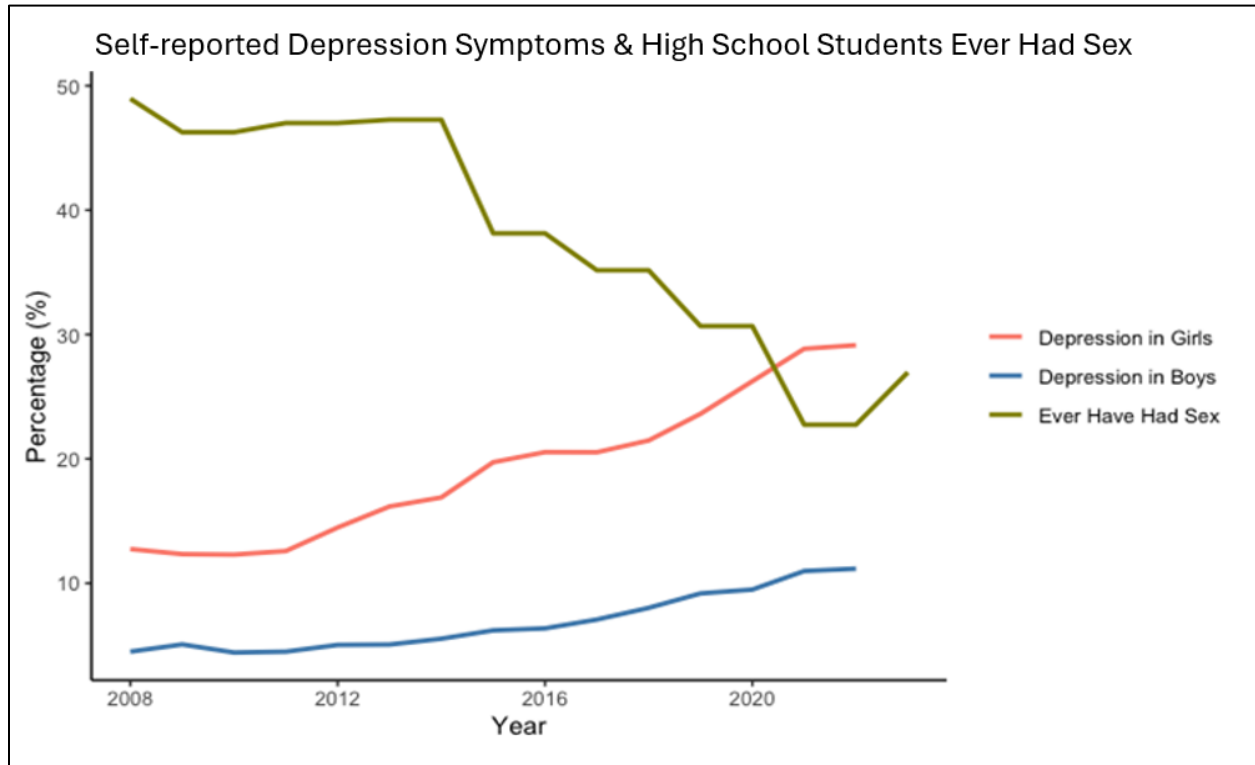


Figure C: plotted using data from the YRBSS

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36. Finally, I looked at other datasets and found a significant correlation between the average number of three-point attempts per team per game in the NBA and the rates of self-reported depression among adolescent girls. I obtained the data from a sports website (Pekmic 2023) and visualize them in Figure D. The naïve Pearson’s correlation between three-point attempts and self-reports of depressive symptoms in girls in the YRBSS data is 0.97 (a score of 1.0 is a *perfect* correlation). In my personal experience as an analyst, that is a shockingly high correlation for real-world, human data, given that these two variables are unrelated. Causation is valuable because it means that we know how to change one variable to facilitate a change in another variable. If correlations were similarly useful then stopping NBA teams from attempting three-pointers would help us address mental health issues in adolescent girls, which obviously would not occur.

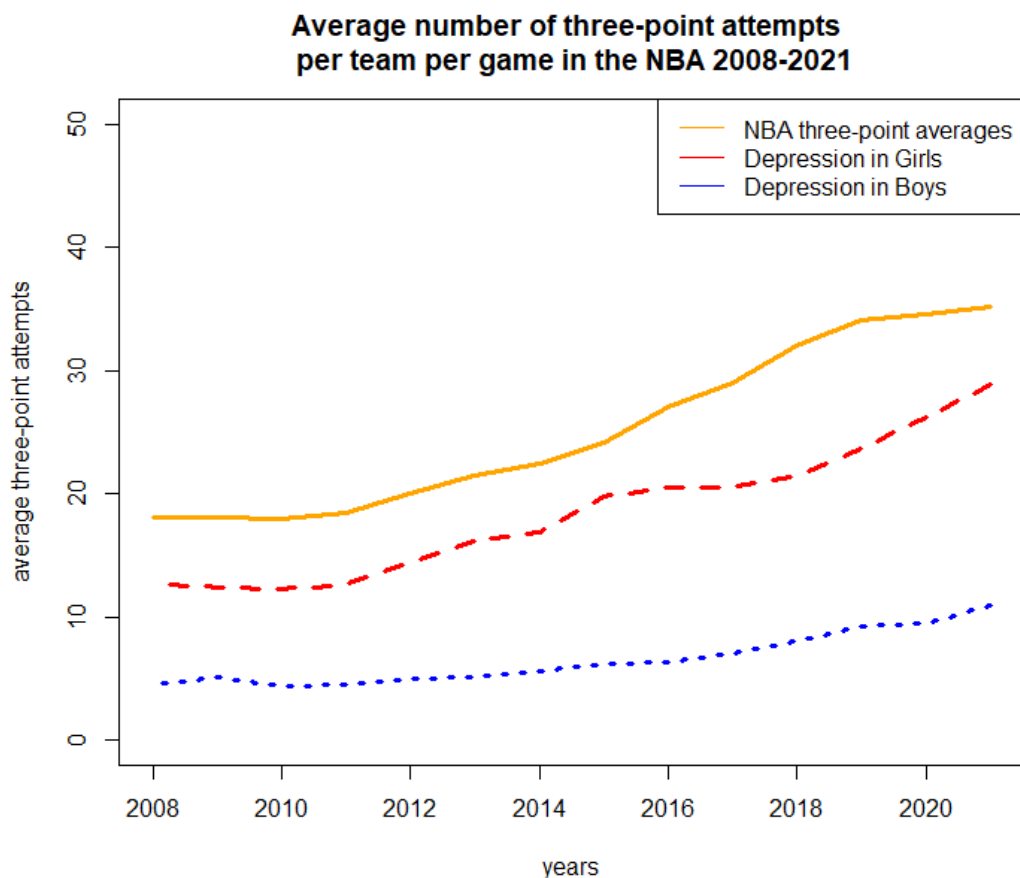


Figure D: The plot summarizes the average number of three-point attempts per game per team in the NBA, and shows its correlation with self-reported rates of depression in adolescent girls and boys as recorded in the YRBSS. Note that the y-axis is only labeled appropriately for the three-point data, but it is scaled appropriately and is *also* correctly interpreted as “% of adolescents reporting depressive symptoms in the YRBSS.”

37. In closing regarding the correlational arguments presented by Professor Twenge, I note that Professor Twenge cites Rohrer (2018)’s “Thinking Clearly About Correlations and Causation: Graphical Causal Models for Observational Data,” a methodological paper outlining

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the challenges of using observational data (i.e., non-experimental data — often collected through surveys or administrative records) to empirically engage causal questions. This manuscript is clear about the serious challenges faced in trying to use observed correlations to make causal claims.

38. I note three quotes from Rohrer (2018) that are relevant to Professor Twenge’s claims about correlation:

- Quote 1: “A widespread approach in the social sciences is to use multiple regression models to achieve statistical control. The dependent variable can be regressed on both the independent variable and the covariate to ‘control for’ the effects of the covariate and thus to potentially block back-door paths. In the standard case, psychologists run models in which linear relationships are assumed without explicit justification. However, this approach does not guarantee adequate adjustment for the covariate.”
 - Comment: Nearly all of the papers using observational data that are cited by Professor Twenge’s report use a version of multiple regression (e.g., generalize linear models, autoregressive moving-average models). As Rohrer points out, these models are not reliable for obtaining causal estimates. These methods make indefensible assumptions (e.g., that no relevant variables were excluded from the model, that all variables in the regression were measured without error, and that the variables are all entered into the model in the mathematically correct manner). Regressions like those used in research cited by Professor Twenge are not recognized by experts as rigorous methods for estimating causal effects because they rely upon unrealistic assumptions.
- Quote 2: “Measurement error can affect all methods of statistical control. For example, intelligence ... cannot be measured perfectly. Thus, the statistical adjustment for intelligence is likely not able to completely remove its confounding influence, and the effect of educational attainment on income might be mistakenly assumed to be stronger than it actually is, as a result of residual confounding.”
 - Comment: As discussed elsewhere in this report, the literature on social media and mental health has demonstrated that its core measurement — i.e., self-reported time using social media — suffers from a pernicious form of measurement error. As the excerpt from Rohrer (2018) suggests, having a biased measurement of the “cause” under consideration is an extremely bad position for an empirical discipline to be in. See Section VI, particularly the discussion about Hunt et al. (2018) which establishes serious issues of recall-bias with measurements of social media usage. Without a reliable measurement of the exposure (i.e., hours using social media), we have no basis for reliable empirical conclusions, let alone causal conclusions.
- Quote 3: “In reality, researchers may often end up with data that do not contain reliable measures of central confounders ... because somebody else collected the data (e.g., they came from a nationally representative panel or survey studies), or because the confounder is some unobservable factor that could not be measured with available methods. In such a

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situation, thorough consideration of the causal web underlying the variables can lead to the conclusion that the data do not warrant causal claims.”

- Comment: Professor Twenge’s report, in its emphasis on correlational and time series studies, does not engage with the serious issues of confounders biasing selection in how social media is being used by adolescents. To be clear: this is a devastating shortcoming. No evaluation attempting to identify a causal effect can be rigorous without an empirical strategy for addressing confounding arising from both observed and unobserved covariates (e.g., randomized trial, natural experiment, formal sensitivity analyses).

39. These are just a handful of the cautionary criticisms offered in Rohrer (2018), and each of these provides a canonical description of why correlational studies are presumed to produce biased results. Using the criteria laid out in Rohrer (2018), when I review the correlational literature cited by Professor Twenge, I judge this correlational literature to be unable to produce reliable causal effects.

d. Professor Twenge’s Discussion of Internal Company Documents Does Not Bolster Her Correlational Claims

40. Professor Twenge cites several Meta internal documents in her report and claims that these materials find similar associations to other studies discussed in her report (Page 20). There are several reasons I outline below why these documents do not rise to the level of evidence that I would consider in a scientific argument.

41. First, the findings Professor Twenge cites from these internal studies are either correlational surveys or qualitative focus groups—neither of which can establish causation. For example, she cites two internal document studies that report that those “who spend more than an hour a day on Instagram are more likely to report negative mood” (Page 20). However, those data were from surveys, making the results correlational and unable to justify causal conclusions. Similarly, the focus groups from which she selects quotes are qualitative and exploratory; they do not appear designed to test hypotheses or establish causal links. Moreover, such focus groups are methodologically limited and vulnerable to multiple forms of bias—including demand characteristics, selection bias, confirmation bias, and lack of replicability.

42. Second, the measures used in these internal studies are not scientifically validated. These studies also rely on participants’ self-reported social media use, which is known to be unreliable. In fact, the same internal report that Professor Twenge cites—claiming that those who spend more than an hour a day on Instagram report more negative mood—explicitly acknowledges that “a majority of people cannot accurately estimate the time they spend on Instagram” when comparing reported versus actual use (META3047MDL-032-00000933). Additionally, these studies assess mental health using vague and unvalidated items such as “negative mood” or whether participants “feel better or worse”; these are not standardized or clinically validated measures of depression, anxiety, or long-term psychological well-being. With many of the studies cited in these internal documents, we do not know what was actually being measured because the metrics are not formally stated.

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43. Third, Professor Twenge’s references to internal documents are inaccurate and misleading. She cites one focus group report as evidence that it “identified a relationship between time spent on social media and eating disorders (META3047MDL-020-00250464),” (pg. 20), but that is not what the document showed — nor was that even the focus of the research. The actual purpose of the focus group was to explore how to design social media features that might reduce disordered eating. The “relationship” to which she refers appears only in the background section of the document (pg. 3 of META3047MDL-020-00250464), where a preliminary, correlational finding from unrelated research is briefly mentioned — not something generated or tested by the focus group itself. This kind of confusion happens more often in non-peer-reviewed research because the reports are not standardized, and they have not gone through careful editorial and reviewer-based improvements for clarity.

44. Most significantly, the Meta documents Professor Twenge cites do not provide the level of evidence that is typically accepted in the academic community as scientifically reliable. I struggled to understand the actual methodologies used to collect and analyze their studies because the write-ups do not exhibit the characteristics I would expect to see in a reliable, published study. These studies are not peer-reviewed. Peer review is a rigorous process that serves as an objective checkpoint in academic literature for ensuring validity or credibility of research findings. Studies that are submitted for publication in the academic literature go through a multi-stage peer-review process where independent experts in the same field read a (typically blinded) copy of the manuscript and provide (typically blinded) reviews of the methodology, data integrity, logical reasoning, relevance, and claims made by the authors. While the degree of quality of peer review depends on the reviewers and the journal, at the very least it is an objective gatekeeper to filter out flawed or unsupported claims and conclusions before contributing to the body of public, published science. Internal industry research is not subjected to independent scrutiny in its design, methodology, or claims.

45. I do not regard these internal document research studies as having passed any objective test of rigor, reliability, or validity. In short, these studies do not provide evidence of any causal connection between social media use and adverse mental health outcomes.

III. Granger-Causality, Which Professor Twenge Relies Upon, Has Minimal Bearing on Causation, and It Is Widely Accepted as Unreliable for Assessing Causation.

46. Professor Twenge refers to her use of Granger Causality in a 2018 article and claims that the study “demonstrated that U.S. smartphone ownership and internet use ... predicted adolescents’ unhappiness ... one year later” and “show[ed] a causal pathway from smartphone adoption and internet use to unhappiness among adolescents.” (Pages 9-10). This causal claim is invalid because Granger causality has been rejected by modern statistics as a form of causal analysis.

47. C.W. Granger introduced his notion of “causality” in 1969, in an era prior to modern empirical causal inference. Though Granger defended his notion of causality, in Granger & Newbold (1986) he acknowledged it was best to think of Granger-causality as “temporally related” (i.e., demonstrating correlations, not causation).

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48. Today, Granger causality is distinguished from actual causation (i.e., causation indicating that manipulating A would then give rise to a change in B) by using the designation Granger-causation or G-causation (*notis bene* the hyphenations). Granger-causation is properly described in the literature as a method for identifying the utility of variable A for predicting variable B (particularly in time series settings). In the empirical literature, prediction is distinct from causation and prediction and causation are only indirectly and tenuously linked to each other (e.g., many variables that are not causally linked can be predictive of one another). Famously, shortly after Granger formalized his notion of causality in 1980, Sheehan & Grieves (1982) published a study demonstrating how the Gross National Product of the United States was empirically Granger-causal with an unrelated phenomenon: the number of sunspots.

49. After the modern framework for causal inference was developed, referred to as the “potential outcome framework,” it became clear mathematically that Granger-causality can easily be “fooled” by falsely attributing causality when it is not there, getting the direction of causality wrong, and missing causality if it is present. A Granger-causality analysis is correlational in nature; it is not a rigorous causal analysis (Holland 1986). In modern texts, authors emphasize that Granger-causality is “not generally informative about causal effects” and is useful only for identifying two time series that are correlated and therefore useful for predicting each other (Shojaie & Fox 2022).

50. The two examples of correlations I introduced previously, Figures B and C, produce similar degrees of “Granger-causality” as the use of social media with depression. The correlation between NBA three-pointers and depression rates summarized in Figure D is even more “Granger-causal.” Granger-causal is an unreliable metric when trying to investigate causal attributions. RCTs are the methodological gold-standard.

51. Other than its use of the word “causal” (a misnomer), Granger-causality shares very little with the kind of causality that is necessary to understand the question at issue in this litigation. Granger-causality is methodological pyrite.

52. Professor Twenge asserts conclusions in her report that make it appear she:

- Is not aware that Granger-causality is not capable of establishing causality.
 - E.g. “This shows a causal pathway from smartphone adoption and internet use to unhappiness among adolescents.” (Twenge, 10)
- Incorrectly believes Granger-causality can determine which time series precedes another.⁹
 - “Thus, smartphone ownership and greater internet use increased first, before the increase in adolescent unhappiness.” (Twenge, 9-10)

⁹ The model cannot determine which time series precedes the other. In fact, the user of the method must input the ordering information into the model. That is, this statement by Professor Twenge confuses how this method works: ordering is an *input* to the model — ordering is **not** an empirically determined *output* of the model.

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- Seems to believe that Granger-causality can rank-order¹⁰ causes.¹¹
 - “The study demonstrated that U.S. smartphone ownership and internet use (one time series) predicted adolescents’ unhappiness (another time series) one year later more strongly than unhappiness predicted smartphone adoption and internet use one year later.” (Twenge, 9)

53. These three assertions Professor Twenge makes in her report are discordant with the underlying research she cites. Indeed, in the Twenge et al. (2018) article Professor Twenge cites (and which she also first-authored), the authors are clear that they are unable to reach conclusions like those discussed above:¹²

First, other variables not assessed here could play a role; second, some assessed variables may play a more complex or indirect role; and third, these are correlational data that cannot definitively uncover causal evidence.

54. As with overstating the strength of correlational studies to determine causality, Professor Twenge’s comments about Granger-causality demonstrate a similar methodological weakness in her report. Neither of these methods or study designs can empirically demonstrate causality, despite Professor Twenge’s claims to the contrary.

¹⁰ Suppose that we are examining two correlations: (i) between time series A and time series B, where the correlation is 0.6, and (ii) between time series A and time series C, where the correlation is 0.2. Note that the first correlation is larger than the second. There is no guarantee that there are causal relationships between A and B or between A and C. Further, suppose we are told that there are causal relationships. It does not then follow that the causal relationship between A and B is stronger than between A and C. Measures of correlation sizes are not determinants of causal effect sizes. This is all because correlations (as we are describing here) are “naïve” and only focus on the two variables at a time, to the exclusion of all other possible variables that could be causing the observed variations.

¹¹ Though Professor Twenge uses the term “predicts” in this quotation, she then concludes the same paragraph with a sentence asserting an established causal pathway from smartphone usage to depression. Her logic then implies that this quoted sentence is actually about establishing causation, rather than prediction, as Granger-causality analyses are the only pseudo-causal techniques deployed in Twenge et al. (2018).

¹² Saying that “other variables not assessed here could play a role” gives away the game; that concession is saying that unobserved variables may be the actual causal factors giving rise to the observed changes in the outcome. The authors are (correctly) conceding that their analyses are insufficiently rigorous to address bias arising from confounding.

HIGHLY CONFIDENTIAL (COMPETITOR)**IV. Time Series Are Not the Same as Natural Experiments, and Natural Experiments Rarely Provide Insight Into Causation**

55. Professor Twenge seriously misrepresents the strength of time series data and their ability to empirically achieve causal inferences. I focus on two quotes from Professor Twenge's report, which are misleading.

56. *First*, in Section 3.1 Time Series Studies, on Page 3, Professor Twenge writes: "Time series studies are sometimes called 'natural experiments' as they can demonstrate the consequences of changes in the environment." This is only true in a very limited way. Natural experiments are observational studies that, in a technical sense, have properties that make the data behave almost as if they came from a randomized trial. Natural experiments are not everyday occurrences, but they do exist. For example, the 2021 Nobel Prize in Economic Sciences was awarded to the pioneering researchers who developed the mathematical, statistical, philosophical, and practical foundations for identifying and analyzing natural experiments. If Professor Twenge's claim were generally true, then we would have many examples of observational studies using time series data that can demonstrate causal effects. In fact, however, we do not have many examples of such studies, relative to the number of time series that exist.

57. As a researcher who has published both methodologies and policy research using natural experiments, my opinion is that Professor Twenge's claim is roughly equivalent to saying the following: "Human beings are sometimes called 'Olympians,' as they demonstrate great feats of strength and skill and can even win gold medals." It is true that being a human is necessary to qualify as a gold-medalist in the Olympic games, but only an extremely small number of humans ever meet the sufficient requirements for being an Olympic athlete. Further, even among Olympic athletes, very few of those go on to achieve the extraordinary achievement of winning a gold medal (equivalent to: "proving a causal connection"). In this context, the proper designation in empirical research for the kind of causality-estimating natural experimental study is an "interrupted time series" (a particular kind of discontinuity design)¹³, and none of the studies cited in section 3.1 of Professor Twenge's report rises to that level of rigor.

58. Of the citations offered by Professor Twenge, Braghieri et al. (2022) (first cited in section 4.1.2 part A, page 9) is worth further consideration here. That study uses the staggered rollout of Facebook at colleges as a type of natural experiment design.¹⁴ This is one of the

¹³ Discontinuity designs take advantage of abrupt changes to estimate causal effects. A common study type in the medical literature observes what happens when a medication is abruptly pulled from the market. Patients who got sick in the "pre" period had access to the medication, but those who got sick slightly later and received care in the "post" period do not have access to it (and likely switched to a different medication). Thus, the timing of when one got sick is like a pseudo-randomizer between the old drug that got pulled and the drug(s) that are now used in the "post" period. We call this a "natural experiment" analysis, which is better than most observational studies at estimating unbiased causal effects.

¹⁴ Though technically they implemented their design, and describe their design, as a "differences-in-differences" study design, Braghieri et al. (2022) make use of some of the

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stronger study designs included in Professor Twenge’s report (Section 4.1.2, Page 9). However, there are issues with Braghieri et al.’s analysis, which significantly limit its value to the current litigation. For example: (i) Braghieri et al. (2022) investigated Facebook’s introduction to campuses in 2005, prior to Facebook’s implementation of many of its modern features like those discussed in Plaintiffs’ Complaint; (ii) the surveys used in Braghieri et al. had low response rates and, given the low response rate, there is significant potential for bias in those who responded; (iii) the study cannot identify the impact of features of social media (e.g., distinct from content, differentiated across features); (iv) the study cannot estimate effects for anything but short-term experience with Facebook (i.e., their study looked at the effects of the very first months of exposure, and cannot inform how someone might experience social media even 9 months or more later — an important distinction because we experience the “novelty” of a stimulus that is different from our habituated experience of a stimulus); and (v) they do not have individual-use information from the participants (i.e., they cannot say if any of the students who responded to the survey actually used Facebook, or how much they used Facebook; they can merely say that it was available), meaning the study is “ecological” (estimating group-level effects) and not “individual” (estimating for study participants), and there is no guarantee that a population-level effect would translate to individual-level effects. Accordingly, the Braghieri et al. (2022) study is open to serious criticism that limits its utility for this litigation. The other observational studies cited by Professor Twenge in her report fall well short of the level of rigor required for establishing causal estimates.

59. *Second*, Professor Twenge offers a misleading characterization of what is necessary for a time series analysis to achieve a rigorous causal inference when she says: “Time series studies can point toward causation if one factor occurs before another.” (Page 3). Professor Twenge is, again, citing a necessary condition for causal inference and leaving unsaid that there are many other conditions required. If one variable temporally preceding another was sufficient to “cause” the variable that follows, then we could claim that Christmas cards cause Christmas.

60. Though Professor Twenge describes time series as a special type of study, differentiated from correlational studies and from Granger-causality studies, this is a nonstandard demarcation because studies that assess Granger-causality are just a type of time series analysis (with barely any more rigor in their reported causal inferences). The time series studies cited in section 3.1 of Professor Twenge’s report are correlational and likely biased and fail to qualify as natural experiments. These studies should not be considered distinct from Granger-causal type studies — which also have significant methodological limitations, as discussed above.

V. Professor Twenge Fails to Address Specific Features and Content of Social Media and the Absence of Literature on Specific Features and Content

61. The studies Professor Twenge discusses do not attempt to identify any potential effects of social media features (e.g., aspects of the design of the platforms), separate from the properties of an interrupted time series (i.e., the abrupt introduction of Facebook to campuses forming a “pre” period and a “post” period) to help reduce (though not eliminate) issues of bias arising from variables they could not adequately adjust for in their analyses.

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effects of content. In other words, every empirical result Professor Twenge cites in her report is confounded with content. This is important because there are critical differences between a social media platform itself, the features of that platform, and the content viewed on or delivered by the social media platform (much of which is not produced or posted by the social media platform itself).

62. All of the correlational studies presented by Twenge, and all of her publications, ask about general use of social media (via self-report, which is inaccurate compared to actual use) but do not detail or control for what content was viewed while using the social media site. The studies do not ask what participants did or viewed while using the social media site. For example, in Twenge et al. (2018), the researchers asked, “how often do you visit social networking sites?” Accordingly, the study did not rigorously identify or measure the features (e.g., “likes,” filters) and content viewed (e.g., penguins or politics). This makes it impossible to estimate, in isolation, the effects that (i) the features of the social media platform, or (ii) the content delivered on the platform might have on adolescents. That is, without careful consideration, it is entirely reasonable to believe that any empirical conclusion reached about *social media platforms* is actually better attributed to the *content* being accessed on social media than the features of social media.

63. The above discussion is not just theoretical. McComb’s 2023 systematic review noted empirical findings that suggest content potentially moderated the (small) correlation between social media and some psychological outcomes. While this was not a causal finding (it was observational), the finding that content was a potential moderator of the effects means it is necessary to consider content as a confounder — or even a primary determinant of — users’ experiences and reported mental health outcomes.

64. It is perhaps unsurprising that Professor Twenge cites relatively few studies on specific features. Very few empirical studies have attempted to analyze features, and those that do try to isolate the effects of features of social media services on mental health outcomes have to negotiate three dynamics that are in tension with each other:

- Experimental studies could in theory be used to estimate the effects of a specific feature. However, when tightly controlling the use of the features (to rigorously estimate the effect of features isolated from the effect of content), an experiment may (depending on its design) subsequently lack “external validity” (i.e., applicability to the world outside the study) because adolescents do not naturally use those features the same way the study forces them to use the features during the study (e.g., clicking on photos manipulated to have more “likes” while inside an fMRI scanner does not translate to natural use; for example: Sherman et al. 2016, Sherman et al. 2018).
- Similarly, experimental studies looking at the impact of social media services on clinical mental health tend to have short durations because it may be unethical to risk causing (or withholding improvement for) mental health issues (Hunt et al. 2018, Allcott et al. 2020, Davis & Goldfield 2024).¹⁵ However, the actual measures of clinical outcomes often

¹⁵ Note that: (i) these three studies are cited by Professor Twenge in her expert report, (ii) these studies were of short duration, intervening and measuring outcomes at the four-week mark

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require longer time horizons. These studies may also be challenging to run logistically (e.g., maintaining adherence to the protocol over several months is costly in person-hours and financially).

- To avoid the challenges imposed by an experimental design, researchers may attempt to use observational data (the study types described in Rohrer (2018)). In observational settings, while certain challenges are lowered, and the external validity of the results could potentially be stronger because the data come from real-world usage, the challenge then becomes that it is may be difficult to determine a way to appropriately isolate the effect of a social media service feature from the effect of the content it is associated with. In other words, if social media users are left to use social media as they naturally would, it will be hard to draw any conclusions about specific aspects of their use (i.e., features vs. content) purely based on observations. Additionally, it is challenging to find a natural experiment that happens to pseudo-randomize two things at the same time (i.e., as relevant here, one pseudo-randomizing feature-usage and another pseudo-randomizing the content) and both pseudo-randomizers happening for the same people.

65. No current empirical study offers a statistically rigorous method for isolating the effects of features independent of content.

66. Because the studies Professor Twenge relies on fail to isolate the effects of features or content viewed by users, and consider only social media use as a whole, Professor Twenge's arguments do not inform the causal question at issue.

VI. A Majority of the Studies Professor Twenge Relies on Use Unreliable Measurements, a Limitation Professor Twenge Does Not Acknowledge or Address.

67. Professor Twenge ignores the limitations of the studies in the empirical literature on social media platforms, which lack reliable measurements of usage of social media services. Having poor measurements of usage is extremely consequential because, if we do not have high-fidelity measurements of usage, then we do not have any real way of identifying a causal effect. As an analogy, imagine trying to understand the effect of a new medication by running a study in which participants can take the medication at any dose, at any time, under any condition, and where they are not required to *accurately* tell the researchers how much they took, when they took it, or under what conditions it was taken. Such measurements would be almost entirely meaningless because they would not account for the vast number of confounders that could affect a person's experience. However, studies of social media use that merely ask how much time participants spent on social media exhibit this exact flaw — and Professor Twenge relies on

or earlier, and (iii) none of these studies are able to isolate and attribute change due to the effect of social media features versus the effects of content. In my review of the literature, I identified many studies that used outcomes measured immediately after the intervention, or within hours. I did not locate studies involving randomized trials that had outcomes measured months later. I note that many mental health disorders in the DSM-V require symptoms to persist for several weeks (e.g., six weeks of consistent depressive symptoms).

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such studies at length (Twenge et al. 2018, Hisler et al. 2020, Rothwell 2023, Twenge et al. 2017, Twenge et al. 2019).

68. Consider “demand characteristics” as a particular challenge to accurate measurement. “Demand characteristics” refers to how participants can often guess the study’s hypothesis and may respond in ways that confirm the study hypotheses. One study (Hunt et al., 2018), which Twenge references as one of the few experimental studies showing an effect of decreasing social media time, demonstrates demand characteristics. The researchers asked students about their mood, asked them to either decrease or not change time on some (but not all) social media platforms, and then asked about their moods again. When asked about their usage using this protocol, participants tended to confirm the study hypothesis as their experience (i.e., using social media less improved their mood). However, a more accurate and objective measurement of their actual social media use had no correlation with their well-being and thus defied the study’s hypothesis. In the more accurate-measurement protocol, instead of having participants *recall* their social media usage at *the end of the week* and at the same time as reporting their mental health, the researchers used the more accurate measurement of asking for contemporaneous reports of social media usage during the week (e.g., daily) and then asking the participants to report their mental health at the end of the week (e.g., the measurements were separated in time). Memories are not perfect. Our recalls are often biased. In the Hunt et al. study, it was clear merely changing *how* they measured social media usage changed the study’s findings.

69. Additionally, measurements such as “hours spent on social media” or “hours spent on screens” are not reliable or granular ways of evaluating actual social media use and are subject to inaccurate recall by study participants, but Twenge fails to address this. Retrospective self-reports of social media use (e.g., “how many hours did you spend on social media last week”) are what is typically measured in social media effects studies, but these measurements are demonstrably inaccurate. Therefore, these measurements are not a valid way to look for effects. In fact, Hunt et al. (2018) demonstrates this inaccuracy. When individuals’ actual usage of social media was considered (using an objective measure of their past 7-day use), the study found *no* correlation with depression, loneliness, anxiety, perceived social support, self-esteem, or well-being. However, when the researchers ran the same correlations with self-reported use, they *did find* a significant negative correlation with perceived social support and a marginal but not significant correlation with self-esteem and overall well-being (Hunt et al., 2018). Hence, the objective measurement found no correlation, while the self-reported measurement did. It is well-established that the effects of an exposure are properly measured based on the *actual exposure*, not a person’s *self-report of their exposure*.

70. In the empirical literature investigating a possible connection between social media usage and mental health outcomes, many of the studies are of poor quality because they rely on hours-used as a measure of social media exposure (Twenge et al. 2018, Wilksch et al. 2020, Riehm et al. 2019, Santos et al. 2023, Hunt et al. 2018). Without carefully tracking what is being done while using social media, it is likely impossible to know what features of the apps or behaviors of the users are causing any observed changes. This is a challenge for both experimental studies and for studies using observational data. This is not a minor limitation of

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this literature; in fact, this limitation means that much of this literature is fundamentally incapable of yielding causal inferences because we do not know what is being studied.

VII. Professor Twenge Fails to Properly Consider Potential Alternative Causes

71. In Section 4.1.2 (“Other Suggested Causes”) Professor Twenge makes several arguments that are deeply flawed. If we were to take her argumentation seriously then we would be forced to (i) reject basic tenets of social psychology, (ii) conclude that social media apps could not possibly be the cause of a change in adolescent mental health, and (iii) believe that doctors do not influence our understanding of health except through direct diagnoses.

72. First, in paragraph 43, page 14, Professor Twenge asserts:

“When assessing possible causes of the increases in poor mental health, it is important to focus on population-level factors rather than individual factors. Time series studies are not concerned with all of the possible causes of poor mental health among individuals; instead, they must concentrate on possible causes that appear among most of the population. Thus, factors that impact only a small number of individuals are unlikely to explain time-series trends. For example, a natural disaster affecting people in one area is unlikely to explain population-level changes in mental health.”

73. This does not pass a test of reasonableness: while I may not have directly experienced wars taking place in Europe and Asia, I can experience emotional changes due to these events. The field of social psychology has well-developed theories that describe how individuals gain experiences, learning, and emotional adaptations based on social interactions (the mini-review by Herrando & Constantinides 2021 cites many, but not all, such theories). These theories have been in the literature for many decades: the triadic theory of reciprocal determinism, social modeling theory, social norm development theory, with luminaries like Albert Bandura articulating many of these (Bandura 1978, Bandura 1991, Bandura 2001). These theories have given rise to empirically validated interventions and examples that have been repeatedly confirmed. Professor Twenge’s assertion that individuals must have direct experiences in order for a causal relationship to exist is in direct conflict with well established tenets of social psychology.

74. Second, in paragraph 45, page 14 Professor Twenge asserts:

“Even if we accept the unproved premise that willingness to admit to symptoms has steadily increased, it still cannot explain the trends. To do so, willingness to admit to symptoms would have had to decline between the early 1990s and 2011 and then suddenly increase after the early 2010s (as in Figure 4 of self-reported symptoms of depression). There is no evidence for such a curvilinear pattern.”

75. This is an example of the single-cause fallacy; this fallacy claims that only one cause can give rise to a change in an outcome and if this single cause cannot explain all the ways that the outcome changes then the proposed cause cannot be a cause. This is nonsense. For most outcomes of interest, there are many causes that bring about change in the outcome. In fact, this

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is why scientists spend so much time and effort as researchers to *isolate* the effect of a particular cause on a particular effect – i.e., there are so many *competing* causes that it becomes “noisy” and we have developed procedures like randomized controlled trials to dampen down the effects of the competing causes. Indeed, this argument is in direct tension with Professor Twenge’s asserted opinion that social media apps are the cause of changes in adolescent mental health. Using Professor Twenge’s single-cause logic:

“Even if we accept the unproved premise that [social media use] ~~willingness to admit to symptoms~~ has steadily increased, it still cannot explain the trends. To do so, [social media apps usage] ~~willingness to admit to symptoms~~ would have had to decline between the early 1990s and 2011 and then suddenly increase after the early 2010s (as in Figure 4 of self-reported symptoms of depression). There is no evidence for such a curvilinear pattern.”¹⁶

76. Third, in paragraph 46, on page 14, Professor Twenge asserts:

“The evidence for the rise in adolescent depression presented in section 4.1 is from nationally representative surveys outside of health care settings. These surveys do not rely on physician diagnoses or physician screening. Thus, factors such as physicians' willingness to diagnose depression, greater physician screening for depression, or changes in diagnostic coding for depression cannot explain the increases in depression or depressive.”

77. This argument does not hold after a moment of thought. Consider: (i) if an adolescent is diagnosed with depression by a physician then the adolescent is more likely to self-report depressive symptoms, (ii) if a physician screens an adolescent for depression then merely by asking them to recall and think about those symptoms it increases the chances the adolescent will notice, recall later, and be able to articulate these symptoms in a self-report, and (iii) even if an adolescent does not interact directly with a physician then in the adolescent’s interactions with other people (peers, mentors, teachers) they may encounter people who have learned about depression and its symptoms from a physician and thus learn how to recognize and communicate about their own symptoms. While these dynamics may feel intuitive there is more behind these; these kinds of social interactions, and social learning, are core dynamics described in social psychology (Bandura 1978, Bandura 1991, Bandura 2001).

78. Beyond the issues with Professor Twenge’s argumentation and logic, the empirical claims in this section are unsound. In both the School Shootings and Covid-19 subsections (Pages 12 and 16) of her report, Professor Twenge acknowledges that causes other than social media may be factors in the self-reported mental health in adolescents (e.g., Professor Twenge acknowledges school shootings are “unlikely to be the sole or primary factor in declining adolescent mental health”). While Professor Twenge acknowledges these other potential causes in her report, I do not see how the empirical literature she cites engages with

¹⁶ This is true because social media apps were used less widely prior to 2010 and largely did not exist before the 2000s, so their usage could not have declined. Note that this quotation has been modified to illustrate the assertion more efficiently.

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these potential confounders; that is, I do not see statistical techniques cited that statistically adjust or control for these alternative causes. Without such techniques in the underlying correlational studies, the standard is to presume that estimates are biased away from the causal effect.

79. This discussion is a useful entry point to the following takeaway: population-level changes are often caused by many different factors, particularly when these changes are occurring over many years. For complex trends over time, such as changes in reported mental health outcomes, it is exceedingly rare that causality is one-dimensional, as Professor Twenge suggests here. Therefore, Professor Twenge's summary dismissals of various potential alternative causes indicate she has not properly considered them as factors that may be substantially contributing to increasing adverse mental health among adolescents during this time period.

80. The multifactorial phenomenon of causation is especially true in the area of mental health. Depression itself has significant diagnostic heterogeneity — to consider it only has one cause (social media use) for an increase in prevalence requires ignoring or suppressing the complex science behind this condition. A person can have up to 227 different symptom patterns that meet the criteria of the DSM-V Major Depressive Disorder, which indicates that there is more than one cause (Zimmermen et al., 2015). This level of causal complexity is similar with suicide and self-harm. “Rather than having a single cause, suicide and self-harm are the result of a complex interplay of several factors that occur throughout the life course, and vary by gender, age, ethnicity, and geography” (Knipe et al., 2022).

81. From a causal inference perspective, the complexity of the depression diagnosis, as well as other mental health conditions and diagnoses, necessitates a rigorous investigation of the rising rates of adolescent mental health conditions that includes a consideration of potential alternative causes. Because complex issues such as mental health tend to have complex origins — it often takes many factors for mental health conditions to arise in a *single person* (e.g., genetic disposition, social environment, experiences during development) — to attribute the change in mental health in a *population of adolescents* to just a single cause would require significant empirical justification. I briefly discuss three potential other population-level dynamics that are largely ignored in the literature on social media.

a. Mental Health Parity and Addiction Equity Act of 2008 (MHPAEA)

82. The first major dynamic, which is noticeably absent in Professor Twenge's report, is the Mental Health Parity and Addiction Equity Act of 2008 (MHPAEA), which was later reinforced by the Affordable Care Act (ACA) of 2010. These Acts constituted a landmark shift in U.S. health insurance law that required that mental health and substance use disorder treatment be covered, and they eliminated higher co-pays, visit limits, and coverage exclusions. Rigorous empirical studies (e.g., studies that use rigorous methods such as interrupted time series and difference-in-differences) show MHPAEA and the ACA significantly increased behavioral health service use and diagnoses, especially among adolescents — and especially those from low to middle income families (particularly rigorous studies: Block et al., 2020, Li & Ma, 2020; other research supporting increased use: Breslau et al., 2020, Walter et al., 2017, Ali et al., 2016; Frank

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et al., 2014). Block et al.'s (2020) Figure E reports the adjusted mean monthly total expenditures for specialty behavioral health plans, demonstrating that MHPAEA was estimated to have an increase on spending on behavioral health services in adolescents (reprinted below).

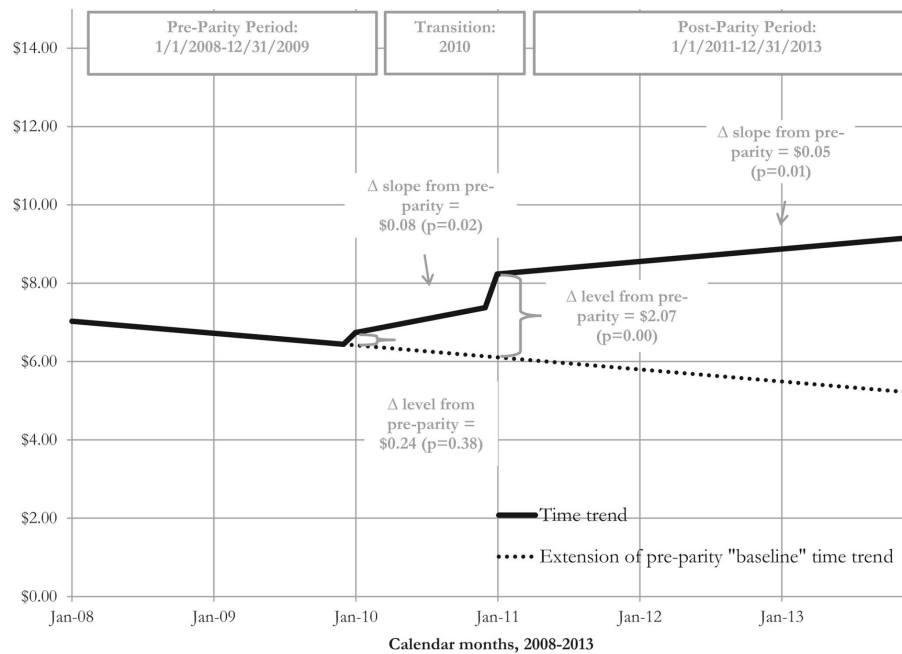


Figure E: This is a reprinted Figure 3 from Block et al. (2020) which reports the adjusted mean monthly total expenditures for specialty behavioral health plans, demonstrating that MHPAEA was estimated to have a significant increase on spending on behavioral health services in adolescents in the 2010 period

83. It is plausible that, before these policies, diagnosis rates and care utilization may have been repressed, and these observed increases in adolescent mental health diagnoses and care utilization may reflect long-overdue access to treatment rather than deterioration in well-being. In fact, that line of reasoning was the contemporary justification for implementing these massive federal policy changes (Frommer 2008, Kirsten et al., Nurden 2024, Frank et al. 2014). Professor Twenge fails to account for this contemporaneous and transformational legislation as an alternative explanation.

84. Related to the above, two other contemporaneous changes impacted the landscape of adolescent mental health diagnoses and service access. First, in 2009, the United States Preventive Services Task Force (USPSTF) began recommending universal screening for Major Depressive Disorder in adolescents (12-18), citing evidence that early detection and treatment could be effective at preventing long term harms (Force USPST 2009). This was reinforced by the American Academy of Pediatrics and the National Association of School Psychologists, in addition to having a mandate for insurance coverage under the ACA (Committee on Psychosocial Aspects Task Force 2009, Psychologists NAOs 2015, National Research Council and Institute of Medicine 2009). These changes expanded access to mental health screening in both primary care and school settings, increasing opportunities for detection and diagnoses.

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85. My point here is not that MHPAEA or changes in diagnostic practices *did cause* increased rates of adverse adolescent mental health outcomes, but rather that Professor Twenge’s failure to properly consider and robustly evaluate these as potential causes undermines her claim that social media is largely responsible for changes in adolescent mental health.

b. Changes in Diagnostic Criteria and Screening Guidelines

86. Second, in 2013, the release of the DSM-5 introduced broader diagnostic criteria for Major Depressive Disorder, with changes such as recognizing “hopelessness” (even without sadness) (Uher et al., 2014) and including bereavement as possible reasons for depression. As a statistician, looking at the expansion criteria for a given diagnosis, I would expect a subsequent increase in diagnoses. Even without other changes in the rate of actual disorders, merely changing how we define and diagnose should change the prevalence of the disorder in our data. Some scholars — notably people directly involved with establishing the new criteria — have noted the possibility of “diagnostic inflation” from the DSM-5 changes, noting potential overdiagnosis and medicalizing normal emotional responses (Dowrick & Frances, 2013).

87. Professor Twenge never empirically addresses these changes in her handling of alternative causes, except to state her belief that these changes could only have had an impact in 2015 and onward because that is when these changes were formally announced. This strikes me as not a very convincing argument for a couple of reasons. First, as discussed in the previous section, there was major legislation occurring in 2008 designed to improve access to mental health care, and there were recommendations in the 2009 United States Prevention Task Force to expand screening around these issues. Second, I do not see statistical methods in the research cited by Professor Twenge that address the potential impact of these population-level changes in mental health care screening and access (e.g., through study design or through statistical adjustments).

c. Gun Violence and School Shootings

88. A third major dynamic possibly contributing to changes in mental health (again, this is not an exhaustive list) is gun violence and school shootings. Professor Twenge briefly considers this as an alternative cause and dismisses it. But if Professor Twenge considers pure correlation as her main argument for causality, then it is not clear why she does not recognize gun violence as a potential factor. She notes that the increase in gun violence, and particularly school-based gun violence, is not perfectly overlapping with the time frame as she defines it (post-2010); however, as noted by the graph below, there was a steep increase in the number of incidents post-Sandy Hook, which occurred in 2012.

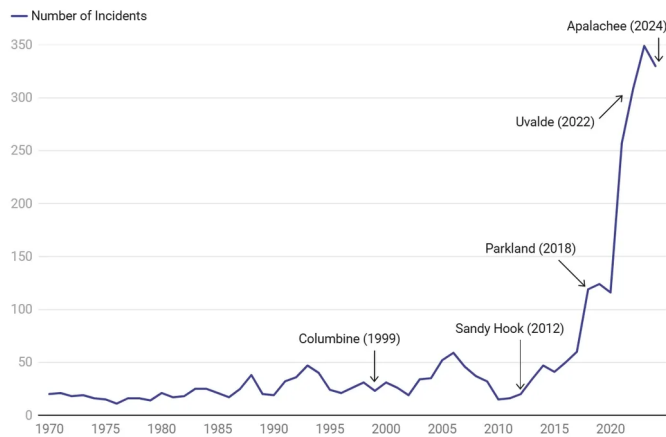
89. There is a clear increase in national, headline-grabbing school violence during the period we are considering. And it is not correct to think that impacts of violence are limited to direct pathways (e.g., an adolescent being involved directly in violence); rather, such impacts likely also work indirectly, including through widespread media coverage, fear induced by school lockdown drills, and general exposure to gun violence in schools (Klemko, 2023; Rapa et al., 2024; Shultz et al., 2013). Studies such as Lowe & Galea (2017) found that mass shootings were associated with adverse psychological outcomes, particularly in vulnerable groups, like

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girls and youths from low-socioeconomic backgrounds, even when they were only indirectly exposed. Additionally, by 2015–2016, 92% of schools had active shooter plans in place (Zhang et al., 2018), and even lockdown drills themselves can induce anxiety and depressive symptoms among students (Riggs et al., 2023; Moore-Petinak et al., 2020).

2024 saw the 2nd highest number of K-12 school shooting incidents

The 2024 count fell only 19 below the all-time high of 349 incidents recorded in 2023



Updated as of Jan. 7, 2025, at 3:30 p.m. ET. The K-12 School Shooting Database defines school shootings as any time a gun is fired or brandished with intent or when a bullet hits school property, regardless of the number of victims, time, day or reason.

Chart: Kara Arundel and Jasmine Ye Han/K-12 Dive • Source: K-12 School Shooting Database • Get the data • Created with Datawrapper

Figure F: reproduced from Rapa et al. 2024 reporting school mass shootings.

90. The direct, indirect, proximal, and distal impacts of school shooting incidents on adolescent mental health are a plausible argument for the increase in depression and mental health disorders. My assessment of the current empirical literature on the impact of school shootings on mental health in adolescents is that it is correlational, and has not established definitive causal relationships. However, the literature on school shootings *does* have enough empirical evidence to warrant being addressed as a potential confounder in any serious analysis of national-level trends in mental health in adolescents.

91. Professor Twenge offers an argument dismissing school shootings:

“In addition, if the primary cause of increases in depression were school shootings, those increases should not appear in countries where school shootings occur at considerably lower rates. For example, the number of young Australians with high levels of psychological distress skyrocketed between 2012 and 2019, in a very similar pattern to the U.S. data (see Figure 5), despite significantly fewer instances of school shootings.”

92. This argument relies on a peculiar assumption—namely, that a person must experience an event directly in order to feel the consequences. That is, if celebrations break out in one part of the world then other regions could not share in that excitement. Humans are behaviorally complex, and citing countries such as Australia and the United Kingdom — which

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are linguistically and culturally overlapping with the United States — seems to minimize the complex ways that culture is shared between communities. Professor Twenge’s dismissal of the potential impact of school shootings on mental health does not take this issue seriously enough as a potential contributor to increased rates of adverse adolescent mental health outcomes.

VIII. Conclusion

93. Professor Twenge’s report fails to apply the proper methods for assessing causality that are widely accepted as the gold standard in the statistics and causal inference community and instead applies unreliable methodologies in making her arguments. She also fails to address the limitations of the studies she discusses, including the inability of correlational studies to establish causation or their failure to estimate the effect of specific features (as distinct from the content being requested by the user). Finally, her analysis of alternative causes is parochial and fails to account for other critical trends emerging in the time period she discusses. For these reasons, Professor Twenge’s core conclusion — i.e., that mental health harms are causally related to social media use — is scientifically flawed and methodologically unreliable.

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Certification

The undersigned hereby certifies their understanding that they owe a primary and overriding duty of candor and professional integrity to help the Court on matters within their expertise and in all submissions to, or testimony before, the Court. The undersigned further certifies that their report and opinions are not being presented for any improper purpose, such as to harass, cause unnecessary delay, or needlessly increase the cost of litigation.

Dated: 7/9/2025

A handwritten signature in black ink, appearing to read "Michael Baiocchi", written over a horizontal line.

Michael Baiocchi, Ph.D.

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HIGHLY CONFIDENTIAL (COMPETITOR)**X. Table S1**

Results of search for systematic reviews and meta-analyses.

First Author	Title	Year
Alfredson QD	Systematic Review of Studies Measuring Social Media Use and Depression, Anxiety, and Psychological Distress in Adolescents: 2018-2020	2024
Alonzo R	Interplay between social media use, sleep quality, and mental health in youth: A systematic review	2021
Arias-de la Torre J	Relationship Between Depression and the Use of Mobile Technologies and Social Media Among Adolescents: Umbrella Review	2020
Berger MN	Social Media Use and Health and Well-being of Lesbian, Gay, Bisexual, Transgender, and Queer Youth: Systematic Review	2022
Blake JA	Will restricting the age of access to social media reduce mental illness in Australian youth?	2025
Blanchard L	Associations between social media, adolescent mental health, and diet: A systematic review	2023
Chochol MD	Social Media and Anxiety in Youth: A Narrative Review and Clinical Update	2023
Choukas-Bradley S	The Perfect Storm: A Developmental-Sociocultural Framework for the Role of Social Media in Adolescent Girls' Body Image Concerns and Mental Health	2022
Course-Choi J	Social Media Use and Adolescent Well-Being: A Narrative Review of Longitudinal Studies	2021
Dibben GO	Adolescents' interactive electronic device use, sleep and mental health: a systematic review of prospective studies	2023
Dienlin T	The impact of digital technology use on adolescent well-being	2020
Draženović M	Impact of Social Media Use on Mental Health within Adolescent and Student Populations during COVID-19 Pandemic: Review	2023
Fassi L	Social Media Use and Internalizing Symptoms in Clinical and Community Adolescent Samples: A Systematic Review and Meta-Analysis	2024
Ferguson CJ	Do social media experiments prove a link with mental health: A methodological and meta-analytic review.	2025
Ghai S	Lack of Sample Diversity in Research on Adolescent Depression and Social Media Use: A Scoping Review and Meta-Analysis	2023
Ghai S	Social media and adolescent well-being in the Global South	2022
Gioia F	Problematic Internet Use and Emotional Dysregulation Among Young People: A Literature Review	2021
Giumetti GW	Cyberbullying via social media and well-being	2022

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Gupta C	Reviewing the Impact of Social Media on the Mental Health of Adolescents and Young Adults	2022
Hamilton JL	Reexamining Social Media and Socioemotional Well-Being Among Adolescents Through the Lens of the COVID-19 Pandemic: A Theoretical Review and Directions for Future Research	2022
Harness J	Social Media Use and Youth Mental Health: Intervention-Focused Future Directions	2023
Hossain MM	Global burden of mental health problems among children and adolescents during COVID-19 pandemic: An umbrella review	2022
Ivie EJ	A meta-analysis of the association between adolescent social media use and depressive symptoms	2020
Kerr B	Associations Between Social Media Use and Anxiety Among Adolescents: A Systematic Review Study	2025
Khalaf AM	The Impact of Social Media on the Mental Health of Adolescents and Young Adults: A Systematic Review	2023
Kostyrka-Allchorne K	Review: Digital experiences and their impact on the lives of adolescents with pre-existing anxiety, depression, eating and nonsuicidal self-injury conditions - a systematic review	2023
Kruzan KP	Social media-based interventions for adolescent and young adult mental health: A scoping review	2022
Liu M	Time Spent on Social Media and Risk of Depression in Adolescents: A Dose-Response Meta-Analysis	2022
Liu Y	Digital intervention in improving the outcomes of mental health among LGBTQ+ youth: a systematic review	2023
Lund L	Electronic media use and sleep in children and adolescents in western countries: a systematic review	2021
Malloy J	Co-design of digital health interventions with young people: A scoping review	2023
Mazzeo SE	Mitigating Harms of Social Media for Adolescent Body Image and Eating Disorders: A Review	2024
McCashin D	Using TikTok for public and youth mental health - A systematic review and content analysis	2023
Moss C	Assessing the impact of Instagram use and deliberate self-harm in adolescents: A scoping review	2023
Odgers CL	Annual Research Review: Adolescent mental health in the digital age: facts, fears, and future directions	2020
Piteo EM	Review: Social networking sites and associations with depressive and anxiety symptoms in children and adolescents - a systematic review	2020
Popat A	Exploring adolescents' perspectives on social media and mental health and well-being - A qualitative literature review	2023

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Prasad S	Anxiety and depression amongst youth as adverse effects of using social media : A Review	2023
Pretorius C	Young People's Online Help-Seeking and Mental Health Difficulties: Systematic Narrative Review	2019
Rajkumar E	Prevalence of mental health problems among rural adolescents in India: A systematic review and meta-analysis	2022
Revranché M	Investigating the relationship between social media use and body image among adolescents: A systematic review	2022
Richards D	Impact of social media on the health of children and young people	2015
Ridout B	The Use of Social Networking Sites in Mental Health Interventions for Young People: Systematic Review	2018
Santos RMS	The associations between screen time and mental health in adolescents: a systematic review	2023
Saiphoo AN	A meta-analytic review of the relationship between social media use and body image disturbance.	2019
Shah J	New age technology and social media: adolescent psychosocial implications and the need for protective measures	2019
Shannon H	Problematic Social Media Use in Adolescents and Young Adults: Systematic Review and Meta-analysis	2022
Taquette SR	Causes and consequences of adolescent dating violence: a systematic review	2019
Throuvala MA	School-based Prevention for Adolescent Internet Addiction: Prevention is the Key. A Systematic Literature Review	2019
Valkenburg PM	Social media use and its impact on adolescent mental health: An umbrella review of the evidence	2022
Vidal C	Social media use and depression in adolescents: a scoping review	2020
Weigle PE	Social Media and Youth Mental Health	2024
Yu DJ	The Impact of Social Media Use on Sleep and Mental Health in Youth: a Scoping Review	2024
Zhou Z	Relationship between online social support and adolescents' mental health: A systematic review and meta-analysis	2022

HIGHLY CONFIDENTIAL (COMPETITOR)**XI. Table S2**

Expanded search for systematic reviews and meta-analysis.

First Author	Title	Year
Ahmed	Social media use, mental health and sleep: A systematic review with meta-analyses.	2024
Ansari	Social media use and well-being: A systematic review and meta-analysis.	2024
Blanchard	Associations between social media, adolescent mental health, and diet: A systematic review.	2023
Brautsch	Digital media use and sleep in late adolescence and young adulthood: A systematic review.	2023
Casale	Has the prevalence of problematic social media use increased over the past seven years and since the start of the COVID-19 pandemic? A meta-analysis of the studies published since the development of the Bergen social media addiction scale.	2023
Cheng	A systematic review and meta-analysis of the relationship between youth drinking, self-posting of alcohol use and other social media engagement (2012–21).	2024
Fassi	Social media use and internalizing symptoms in clinical and community adolescent samples: A systematic review and meta-analysis.	2024
Ghai	Lack of sample diversity in research on adolescent depression and social media use: A scoping review and meta-analysis.	2023
Godard	Are active and passive social media use related to mental health, wellbeing, and social support outcomes? A meta-analysis of 141 studies.	2024
Khalaf	The impact of social media on the mental health of adolescents and young adults: a systematic review.	2023
Khetawat	Examining the association between digital stress components and psychological wellbeing: a meta-analysis.	2023
Liu	Am I Happier Without You? Social Media Detox and Well-Being: A Meta-Analysis of Randomized Controlled Trials.	2025
Liu	The Impact of Social Media on Children’s Mental Health: A Systematic Scoping Review.	2024
Marciano	Does social media use make us happy? A meta-analysis on social media and positive well-being outcomes.	2024

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McGovern	The associations between photo-editing and body concerns among females: A systematic review.	2022
Meynadier	Lower mindfulness is associated with problematic social media use: A meta-analysis.	2024
Meynadier	Meta-analysis of associations between five-factor personality traits and problematic social media use.	2024
Plackett	The impact of social media use interventions on mental well-being: systematic review.	2023
Purba	Social media use and health risk behaviours in young people: systematic review and meta-analysis.	2023
Senekal	Social media and adolescent psychosocial development: a systematic review.	2023
Susi	Research review: Viewing self-harm images on the internet and social media platforms: Systematic review of the impact and associated psychological mechanisms.	2023
Wu	Social anxiety and problematic social media use: A systematic review and meta-analysis.	2024
Yigiter	The relationship between problematic social media use and depression: A meta-analysis study.	2024

HIGHLY CONFIDENTIAL (COMPETITOR)**XII. Table S3**

Labeling of Professor Twenge's list of citations.

First Author	Title	Label
Allcott, H	The welfare effects of social media	Causal Claims
Blanchflower, D	Is the Gallup World Poll reliable? After Babel Substack	Non-Peer-Reviewed
Boer, M	National-level schoolwork pressure, family structure, internet use, and obesity as drivers of time trends in adolescent psychological complaints between 2002 and 2018	No Causal Claim
Boers, E	Association of Screen Time and Depression in Adolescence	No Causal Claim
Braghieri, L	Social Media and Mental Health	Causal Claims
Burstein, B	Suicidal attempts and ideation among children and adolescents in US emergency departments, 2007-2015	Other
Cairns, R	Trends in self-poisoning and psychotropic drug use in people aged 5-19 years: a population-based retrospective cohort study in Australia	Other
Corredor-Waldron, A	To What Extent Are Trends in Teen Mental Health Driven by Changes in Reporting? The Example of Suicide-Related Hospital Visits	Other
Coyne, S	Does time spent using social media impact mental health?: An eight year longitudinal study	Incorrect Claim of Causal Design
Cybulski, L	Temporal trends in annual incidence rates for psychiatric disorders and selfharm among children and adolescents in the UK, 2003–2018	Other
Davis, CG	Limiting social media use decreases depression, anxiety, and fear of missing out in youth with emotional distress: A randomized controlled trial.	Causal Claims
Deters, F	Does Posting Facebook Status Updates Increase or Decrease Loneliness? An Online Social Networking Experiment.	Causal Claims
Faverio, M	Teens, social media, & technology 2024	Other
Ferguson, C. J.	Do social media experiments prove a link with mental health: A methodological and meta-analytic review	Causal Claims
Gajdics, J	Mobile Phones in Schools: With or Without you? Comparison of Students' Anxiety Level and Class Engagement After Regular and Mobile-Free School Days.	No Causal Claim
Gray, P	Decline in independent activity as a cause of decline in children's mental well-being: Summary of the evidence	Other
Gumas, ED	U.S. Overdose Deaths Remain Higher Than in Other Countries — How Harm Reduction Programs Could Help.	Non-Peer-Reviewed

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Gutierrez, C	Type of household firearm ownership and firearm suicide among adolescents	Other
Haidt, J	The Anxious Generation	Non-Peer-Reviewed
Haidt, J	Social media and mental health: A collaborative review	Non-Peer-Reviewed
Heffer, T	The longitudinal association between social-media use and depressive symptoms among adolescents and young adults: An empirical reply to Twenge et al (2018)	Incorrect Claim of Causal Design
Hill AB	The Environment and Disease: Association or Causation?	Other
Hisler, G	Associations between screen time and short sleep duration among adolescents varies by media type: Evidence from a cohort study.	No Causal Claim
Hunt, MG	No more FOMO: Limiting social media decreases loneliness and depression	Causal Claims
Kannan, VD	US trends in social isolation, social engagement, and companionship – nationally and by age, sex, race/ethnicity, family income, and work hours, 2003-2020	Other
Kelly, Y	Social Media Use and Adolescent Mental Health: Findings from the UK millennium cohort	Other
Keyes, KM	Recent increases in depressive symptoms among US adolescents: Trends from 1991 to 2018	Other
Klerman, G	Increasing rates of depression	No Causal Claim
Krokstad, S	Divergent decennial trends in mental health according to age reveal poorer mental health for young people: Repeated cross-sectional population-based surveys from the HUNT Study	Other
Kross, E	Facebook use predicts declines in subjective well-being in young adults.	No Causal Claim
Lenhart, A	Teens, social media, & technology overview 2015	Other
Lepp, A.	The experimental effect of social media use, treadmill walking, studying, and a control condition on positive and negative affect in college students.	Causal Claims
Lewinsohn, P	Age-cohort changes in the lifetime occurrence of depression and other mental disorders.	No Causal Claim
Liu, M	Time spent on social media and risk of depression in adolescents: A dose-response metaanalysis.	No Causal Claim
Madden, M	Teens and technology 2013	Other
Maza, MT	Association of habitual checking behaviors on social media with longitudinal functional brain development.	No Causal Claim
Meshi, D	Nucleus accumbens response to gains in reputation for the self relative to gains for others predicts social media use.	Causal Claims
Miech, R	National Survey Results on Drug Use, 1975-2023: Secondary School Students	Other

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Mitev, K	Social media use only helps, and does not harm, daily interactions and well-being	Causal Claims
Mojtabai, R	National trends in the prevalence and treatment of depression in adolescents and young adults	Other
Newsom, C	Changes in adolescent response patterns on the MMPI/MMPI--a across four decades.	Other
Orben, A	Teenagers, screens and social media: A narrative review of reviews and key studies.	No Causal Claim
Orben, A	The association between adolescent well-being and digital technology use.	No Causal Claim
Orben, A	Windows of developmental sensitivity to social media.	No Causal Claim
Patalay, P	Changes in millennial adolescent mental health and healthrelated behaviours over 10 years: A population cohort comparison study	Other
Plemmons, G	Hospitalization for suicide ideation or attempt: 2008–2015	Other
Przybylski, AK	Does taking a short break from social media have a positive effect on well-being? Evidence from three preregistered field experiments.	Causal Claims
Rausch, Z	The Youth Mental Health Crisis is International, Unless You Rely on a Flawed International Dataset (The GBD)	Non-Peer-Reviewed
Rausch, Z	The fundamental flaws of the only meta-analysis of social media reduction experiments (and why it matters), part 2.	Non-Peer-Reviewed
Reuben, A	Association of childhood blood lead levels with cognitive function and socioeconomic status at age 38 years and with IQ change and socioeconomic mobility between childhood and adulthood	Other
Riehm, KE	Associations between time spent using social media and internalizing and externalizing problems among US youth.	No Causal Claim
Rodman, AM	Development of self-protective biases in response to social evaluative feedback	Other
Rohrer, JM	Thinking clearly about correlations and causation: Graphical causal models for observational data	Other
Rothwell, J	Teens spend average of 4.8 hours on social media per day.	Non-Peer-Reviewed
Saiphoo, AN	A meta-analytic review of the relationship between social media use and body image disturbance	No Causal Claim
Schisterman, EF	Overadjustment bias and unnecessary adjustment in epidemiologic studies	Other
Schmidt-Persson, J	Screen media use and mental health of children and adolescents: A secondary analysis of a randomized clinical trial	Causal Claims

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Schrijvers, K	Three decades of adolescent health: Unveiling global trends across 41 countries in psychological and somatic complaints (1994–2022)	Other
Semken, C	Specification analysis for technology use and teenager wellbeing: Statistical validity and a Bayesian proposal	Other
Spiller, HA	Sex- and age-specific increases in suicide attempts by self-poisoning in the United States among youth and young adults from 2000 to 2018	Other
Stein, D	The Fundamental Flaws of The Only Meta-Analysis of Social Media Reduction Experiments (And Why It Matters), Part 3	Non-Peer-Reviewed
Swanson, S	Firearm access and adolescent suicide risk: toward a clearer understanding of effect size	Other
Thrul, J	Social media reduction or abstinence interventions are providing mental health benefits – reanalysis of a published meta-analysis.	Causal Claims
Twenge, J	Time period and birth cohort differences in depressive symptoms in the U.S., 1982–2013.	Other
Twenge, J	Have some teens benefited in the era of social media?	Non-Peer-Reviewed
Twenge, J	The Age of Anxiety? Birth Cohort Change in Anxiety and Neuroticism, 1952-1993	Other
Twenge, J	Increases in depressive symptoms, suicide-related outcomes, and suicide rates among US adolescents after 2010 and links to increased new media screen time.	Incorrect Claim of Causal Design
Twenge, JM	Academic Pressure Cannot Explain the Mental Illness Epidemic	Non-Peer-Reviewed
Twenge, JM	Here are 13 other explanations for the adolescent mental health crisis. None of them work	Non-Peer-Reviewed
Twenge, JM	This group is more likely to be depressed and think about suicide. Generation Tech Substack.	Non-Peer-Reviewed
Twenge, JM	Parent drug overdoses: The true cause of the adolescent mental health crisis?	Non-Peer-Reviewed
Twenge, JM	The homework bubble has popped	Non-Peer-Reviewed
Twenge, JM	Changes in Parents' Mental Health Did Not Drive the Adolescent Mental Health Crisis	Non-Peer-Reviewed
Twenge, JM	Generations: The Real Differences between Gen Z, Millennials, Gen X, Boomers and Silents—and What They Mean for America's Future	Non-Peer-Reviewed
Twenge, JM	The pandemic was bad for teen mental health. The smartphone and social media were worse	Non-Peer-Reviewed
Twenge, JM	For teens, the loneliness epidemic is not a myth	Non-Peer-Reviewed

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Twenge, JM	Not all screen time is created equal: Associations with mental health vary by activity and gender	No Causal Claim
Twenge, JM	Underestimating digital media harm	No Causal Claim
Twenge, JM	Worldwide increases in adolescent loneliness	Other
Twenge, JM	Specification curve analysis shows that social media use is linked to poor mental health, especially among girls	No Causal Claim
Twenge, JM	Linear correlation is insufficient as the sole measure of associations: The case of technology use and mental health	No Causal Claim
Twenge, JM	Decreases in self-reported sleep duration among U.S. adolescents 2009-2015 and links to new media screen time	No Causal Claim
Twenge, JM	Decreases in psychological well-being among American adolescents after 2012 and links to screen time during the rise of smartphone technology	No Causal Claim
Twenge, JM	Gender differences in associations between digital media use and psychological well-being: Evidence from three large datasets	No Causal Claim
Twenge, JM	Age, period, and cohort trends in mood disorder indicators and suicide-related outcomes in a nationally representative dataset, 2005-2017	Other
Twenge, JM	Trends in U.S. adolescents' media use, 1976-2016: The rise of digital media, the decline of TV, and the (near) demise of print	Other
Twenge, JM	Less in-person social interaction with peers among U.S. adolescents in the 21st century and links to loneliness	Other
Twenge, JM	The decline in adult activities among U.S. adolescents, 1976–2016	Other
Van der Wal, A	Social media use leads to negative mental health outcomes for most adolescents (preprint manuscript)	No Causal Claim
Vázquez-Vázquez, A	Admissions to paediatric medical wards with a primary mental health diagnosis: a systematic review of the literature	Other
Verduyn, P	Passive Facebook usage undermines affective well-being: Experimental and longitudinal evidence	Causal Claims
Viner, RM	Roles of cyberbullying, sleep, and physical activity in mediating the effects of social media use on mental health and wellbeing among young people in England: a secondary analysis of longitudinal data	Incorrect Claim of Causal Design
Vuorre, M	Estimating the association between Facebook adoption and well-being in 72 countries	No Causal Claim
Vuorre, M	Global well-being and mental health in the Internet age	No Causal Claim
Vuorre, M	A multiverse analysis of the associations between internet use and well-being	Incorrect Claim of Causal Design

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Ward, JL	Admission to acute medical wards for mental health concerns among children and young people in England from 2012 to 2022: A cohort study	Other
Wells, G	Facebook knows Instagram is toxic for teen girls, company documents show.	Non-Peer-Reviewed
Zhao, Y	Brain structural covariation linked to screen media activity and externalizing behaviors in children	No Causal Claim
Zhao, Y	Brain structural co-development is associated with internalizing symptoms two years later in the ABCD cohort	No Causal Claim

HIGHLY CONFIDENTIAL (COMPETITOR)**Exhibit A. Literature Review**

I conducted a literature review to identify systematic reviews and meta-analyses of the empirical literature of social media service use and mental health in adolescents. The results of this literature review inform my opinions throughout this report. I summarize my key choices in constructing the review of the literature below.

Systematic Reviews and Meta-Analysis*Eligibility Criteria*

Studies were eligible for inclusion if they meet the following criteria:

- a. Included adolescent participants between (and including) age 10 and 19; and
- b. Examined features of social media services
- c. Were peer-reviewed systematic reviews or meta-analyses
- d. Were published in 2019 or later

Information Sources

I conducted a preliminary search in PubMed. I conducted a secondary review in Google Scholar, as PubMed did not recognize some terms (e.g., “infinite scrolling”).

Search Strategy

The search terms are listed below by category:

1. Population: “adolescent”, “teen”, “teenager”, “youth”, “minor”, “young adult”, “high school student”, “middle school student”; “U.S.” or “United States” to limit the scope
2. Social Media Platform: “social media”, “Snap”, “Snapchat”, “social networking site”, “Facebook”, “Instagram”, “TikTok”
 1. I removed YouTube and Twitter as the studies on these platforms did not appear to inform the litigation because the literature was using YouTube and Twitter as platforms to deliver intervention and not assessing the impact of YouTube and Twitter on people. Therefore, keeping these articles within the search would dilute the usefulness of the resulting set.
3. Features: “likes”, “infinite scrolling”, “photo enhancement”
4. Study design: “review” (“systematic review” or “meta-analysis”)

To perform searches in the above-named databases, these terms were combined using Boolean operators (AND, OR). For instance, the combined search string used in PubMed was:

("adolescent" OR "teen" OR "teenager" OR "youth" OR "minor" OR "Young adult" OR "high school student" OR "middle school student") AND ("U.S." OR "United States") AND ("social media" OR "Facebook" OR "Instagram" OR "Snap" OR "Snapchat" OR "YouTube" OR "TikTok") AND ("likes" OR "infinite scrolling" OR "photo enhancement") AND (Review[Publication Type])

The results of this search can be seen in Table S1, 54 citations.

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Manual Google Scholar Search

To supplement the search for research specifically on features, I augmented the above search in two ways.

Because the PubMed search did not return some phrases (e.g., “infinite scrolling”) to ensure I was not missing any work on this feature, I conducted a manual search for infinite scrolling, social media, and adolescents using Google Scholar.

Search Strategy

The search terms were: adolescent AND infinite scrolling social media

I reviewed the first three pages (30 results) to identify the non-duplicate, primary sources on infinite scrolling. The results of this manual search are in Table S2, 5 citations.

Second, I conducted a manual search for studies on features such as: likes, photo enhancements, social media, and adolescents, published any year, using Google Scholar.

Search Strategy

The search terms for these two searches were: 1) adolescent AND likes social media; 2) adolescent AND photo enhancement digital enhancement social media.

The results from this manual search are also recorded in Table S2, 18 citations.